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OF THE
ROYAL SOCIETY OF MEDICINE

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JOHN NACHBAR, M.A., M.D.
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THE EDITORIAL COMMITTEE

VOLUME THE THIRD

SESSION 1909-10

PART III

ODONTOLOGICAL SECTION	OTOLOGICAL SECTION
PATHOLOGICAL SECTION	SURGICAL SECTION
THERAPEUTICAL AND PHARMACOLOGICAL SECTION	

INDEX



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PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

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COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

ODONTOLOGICAL SECTION



LONDON
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ODONTOLOGICAL SECTION.

CONTENTS.

October 25, 1909.

	PAGE
The Relation of Dentistry to other Branches of Medicine and its Bearing upon the Public Health (Presidential Address). By WILLIAM HERN, M.R.C.S., L.D.S.	1
Infective Disease of the Jaws associated with the Absorption of the Teeth. By A. HOPEWELL-SMITH, M.R.C.S., L.D.S.	9
By F. COLEMAN, M.R.C.S., L.D.S. :—	
(1) Papillomata of the Uvula	20
(2) Exostoses of the Mandible	21

November 22, 1909.

Some Observations on the Bacteriology of Pyorrhœa Alveolaris, and the Treatment of the Disease by Bacterial Vaccines. By J. W. EYRE, M.D., and J. LEWIN PAYNE, M.R.C.S., L.D.S.	29
Unilateral Overgrowth of the Lower Jaw. By STANLEY BOYD, F.R.C.S. ...	47
Fracture of the Mandible set with a Silver Splint made by the Casting Process. By C. SCHELLING, L.D.S.	51

January 24, 1910.

The Vaccine Treatment of Early Cases of Pyorrhœa Alveolaris. By KENNETH W. GOADBY, M.R.C.S., L.D.S.	55
The Bacteriology of Pyorrhœa Alveolaris and the Results of Treatment by Bacterial Vaccines. (Discussion)	61

February 28, 1910.

Some Experiments on the Relative Susceptibility of Different Teeth to Dental Caries. By STANLEY P. MUMMERY, M.R.C.S., L.D.S.	71
By CHARLES A. CLARK, L.D.S. :—	
(1) A Sterilizable Film-holder	85
(2) A Method of ascertaining the Relative Position of Unerupted Teeth by means of Film Radiographs	87

April 4, 1910.

	PAGE
By W. DE C. PRIDEAUX, L.D.S. :—	
(1) Rupture of a Gastric Ulcer while in a Dental Chair	91
(2) Vertical Fracture through the Mandible and Canine Tooth	92
Observations on the Condition of the Mouth in 1,000 consecutive Cases of Chronic Disease. By R. ACKERLEY, M.B.	94

April 25, 1910.

By CARL SCHELLING, L.D.S. :—	
(1) Two Cases of Suppuration in the Maxilla	113
(2) Neuralgia due to Impacted Lower Wisdom Teeth, in which Skiagrams helped the Diagnosis	114
Two Cases of Suppurating Dental Cysts invading the Maxillary Antrum. By HERBERT TILLEY, F.R.C.S.	115
Painful Attrition. By WILLIAM RUSHTON, L.D.S.	118
Two Odontoceles, and some other Cysts. By A. HOPEWELL-SMITH, M.R.C.S., L.D.S.	121

May 23, 1910.

Report of the Honorary Curator. By J. F. COLYER, M.R.C.S., L.D.S.	143
The Spindles of von Ebner. By DOUGLAS E. CAUSH, L.D.S.	144

June 27, 1910.

Some Egyptian Skulls. By ARTHUR S. UNDERWOOD, M.R.C.S., L.D.S.... ..	153
A Case showing the Result of Extraction of the Six-year-old Molars. By GEORGE THOMSON, L.D.S.	156
Discussion on Mr. Hopewell-Smith's Paper on "Two Odontoceles and some other Cysts"	158
Mr. F. J. BENNETT (p. 158)—Mr. WARWICK JAMES (p. 159)—Mr. GABELL (p. 160)—Mr. HOPEWELL-SMITH (p. 160).	

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Odontological Section.

October 25, 1909.

Mr. WILLIAM HERN, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

The Relation of Dentistry to other Branches of Medicine and its Bearing upon the Public Health.

IN considering to what subjects I should devote attention in the course of my address to you this evening, it has occurred to me that I could not do better than say a few words on the relation of dental surgery to other branches of the healing art, and on the very important bearing of dentistry upon the public health. In early times, when the principal work of the dental specialist was the extraction of decayed and offending teeth, these matters doubtless seemed to offer but little scope for prolonged discussion. The immediate removal of the disturber of the bodily weal, like the prompt banishment or decapitation by some Eastern tyrant of unpleasantly obtrusive courtiers, was held to be a procedure based on sound surgical principles that must in all cases be adopted. It saved the patient from a continuation of his suffering and its consequences in medical treatment; also it afforded the operator who might happen to be fairly skilful an easy escape from all responsibility.

What was the cause of decay in the tooth, what the loss of teeth might mean to a patient, and how far his general health had been affected by their disease were not considerations that seemed to invite reflection. The awkwardness of the professional situation when too hasty an application of the forceps had resulted in the drawing of the wrong tooth could always be remedied by the subsequent extraction of the real offender. In the good old days the condition of the teeth of the inhabitants of civilized Europe, so long as it did not entail suffering,

2 Hern: *Relation of Dentistry to other Branches of Medicine*

was a matter of small import except from an æsthetic point of view; and at the present time the equanimity with which members of the lower classes part with tooth after tooth that might by proper treatment have been preserved for years is a matter for amazement. The vast majority of the poor have even less respect for the welfare of their teeth than have those savage tribes that file and drill them in obedience to the dictates of custom.

During the past century the medical profession has gradually come to perceive very clearly that there subsists an intimate relation betwixt the well-being of the teeth and that of the body as a whole, and, on the other hand, that bodily disease and degeneration may exercise a considerable influence upon the condition of the teeth. Their teaching, unfortunately, has been slow in obtaining recognition, and much still remains to be effected. It is a striking fact, as Dr. Newsholme has pointed out, that, owing to the progress of pathological science and the self-denying labours of medical men, the regulations for the public health during the past half-century have resulted, despite increased urbanization, in a decrease in the death-rate of 28 per cent. Public legislation has set up safeguards that have considerably lessened the dangers incurred by operatives in certain manufactures. It is scarcely necessary, nowadays, to point out to the laity what are the injurious effects on the teeth and jaws of poisoning by mercury and phosphorus. The ethical creed of the public conscience no longer countenances the degeneration and misery of groups of human beings for the aggrandizement of capitalists. It recognizes that what Huxley calls "the cosmic process"—i.e., the treading down of the weaker by the strongest and most self-assertive—must in these days give place to "the ethical process," which makes for the survival, not of those that may happen to be the fittest in respect of the whole of the conditions that obtain, but the survival of the ethically best. Enactments have been passed to prevent the adulteration of food, and great care has been taken to insure that water-supplies shall be pure and unpolluted. Much has been done for the commonweal by the giving of instruction, either publicly or in schools, in the important art of cooking. Obviously, increased assimilability of food is even more important to the consumer than a mere increase in the supply. But food of the best quality, and cooked to perfection, may, like poorly invested capital, yield very small returns to the individual who, possessing functionally faulty teeth, can only imperfectly masticate it, and who therefore suffers from disordered digestion and consequent mal-assimilation. His undigested food is as much lost as if it were simply cast

directly into the drains. Further, it may be remarked that the purity of food or of drinking water can be of small avail to the person who pollutes all that he eats or drinks by taking it into a mouth swarming with septic organisms.

It is evident that what has not yet been at all effectually impressed upon the community at large is the interdependence of the state of the mouth and teeth and that of the other organs of the body. Many persons suffer more or less for years before to their surprise they learn, when as a last resource the advice of a medical man or dental surgeon is obtained, that dental incompetency is the evil by which their general health is being seriously affected. All of you must be familiar with the case of the poor debilitated labourer who comes into hospital a complete wreck from malnutrition due to indigestion and defective assimilation of improperly masticated food, his teeth having long ceased to be more than vestiges of their original selves. An allied type of patient not infrequently met with is the pale, anæmic girl, ill-nourished, and unable to benefit by her food because of chronic gastritis or gastric ulcer, for the misery attendant on which she can assign no cause but a malignant Providence that vents its spleen especially upon the defenceless and weakly. Such a patient will often be found to be suffering from the ravages of dental caries to such an extent that scarcely a whole tooth remains to her. Moreover, the cavities in her teeth are serving as foci for the propagation of harmful organisms. How often, again, do we come upon instances of acute or chronic malaise, in which oral sepsis, and not, perhaps, deficiency of teeth, is the crying evil! Among such is the spare, debilitated, sallow-faced female who complains of headache, languor, loss of appetite, flatulence, indigestion, depression of spirits, and sundry other ailments—symptoms that prove to be wholly due to fermentative and putrefactive processes set up in the gastro-intestinal tract by the continual swallowing of pus proceeding from inflamed gums and peridental sacs. A still more serious result of pyorrhœa is septic absorption, setting up toxic infection that frequently eventuates in such grave conditions as empyema, endocarditis, neuritis, osteomyelitis, pyæmia, and septicæmia.

The intimate relationship of dental surgery to other branches of the healing art is realized only when one passes in review cases such as those just referred to, in which it has been impossible to regard the teeth apart from general anatomical, physiological, and pathological considerations. The bearing of septic conditions of the mouth on medical practice is a subject on which it is almost unnecessary for me

4 *Hern : Relation of Dentistry to other Branches of Medicine*

to enlarge. Few, if any, acquainted with the pathology of oral sepsis will be inclined to question the statements of Dr. William Hunter that it is more important as a potential disease factor than any other source of sepsis in the body, and that it forms the chief link between dental pathology on the one hand and medicine and surgery on the other. In our general hospitals we find ophthalmic surgeons associating iritis, choroiditis, iridocyclitis, keratitis-punctata, scleritis—in fact, localized inflammation in any of the vascular parts of the eye—with pyorrhœa alveolaris; laryngologists ascribing laryngitis, pharyngitis, tonsillitis and inflammation in the nasal and maxillary sinuses to pyogenic organisms born and bred in the mouth; aural surgeons insisting on the frequency with which suppurative inflammation of the middle ear is associated with similar inflammatory processes in the mouth cavity; dermatologists assigning acne rosacea and other eruptive affections of the skin to the swallowing of organisms to be found in an unhealthy mouth, and general surgeons associating oral sepsis and appendicitis as cause and effect.

The bearing of odontology on general surgical practice is still more close and clear. The causation of neuralgia of the fifth nerve by pathological conditions of the pulps of teeth is an event of nearly every-day occurrence in the experience of the dental and the general surgeon. Obstinate cases of this nature that have long defied the latter may often be cured by his dental colleague within a few hours by the destruction of an inflamed pulp. One instance within my recollection had occasioned no little anxiety to a medical man for several months, and the patient was ordered abroad for cure, her condition being attributed to the dampness of the English climate. Like many another sufferer, she would have been dispatched to a distant region, and would have carried with her, unremedied, the cause of her trouble, had there been no co-operation between the medical and the dental practitioner. How frequently, again, is collaboration between the surgeon and the dentist necessary in the treatment of alveolar abscesses? The former perhaps lances them, only to find a recurrence of the swelling after a short interval, whilst the latter is enabled to treat the cause. Many a time the boy brought to the surgeon on account of enlargement of glands in his neck will be found to owe his condition entirely to the presence of a septic tooth or root of tooth in his jaw.

The patient who is sent to the general hospital suffering from a sinus discharging on to his face or neck, and who has been told that he will need an operation for the removal of dead bone, may perhaps be

cured by no more serious procedure than the removal of the buried root of a dead tooth. Similarly, patients who come complaining of chronic ulcer of the tongue may often be relieved simply by the removal of a rough root or a jagged tooth. Again, the cause of a large swelling in the jaw may be found to be due to an odontome or to some other aberration of a tooth-germ.

Having endeavoured to show how close and intimate are the relations of dental surgery to other departments of medicine, I will now refer briefly to what I conceive to be our duty to the public respecting the prevention of dental disease. The Interdepartmental Committee on Physical Deterioration drew attention to the fact that a very large amount of physical disability and ill-health results from dental disease. The finding of this Committee is corroborated by the Reports issued by the Education Committee of the London County Council and by the medical officer to that body. Investigations by members of the British Dental Association and other observers show that 80 to 90 per cent. of school children are sufferers from the effects of dental caries. In an elementary school decay of the permanent teeth was discovered to exist in fully one-third of the children between six and nine years of age, and in four-fifths of those between ten and eleven. Inquiries in this country have proved that not only have nine out of every ten children decayed teeth, but about two-thirds of the entire population are suffering in a greater or less degree from dental disease. Dental caries, in short, as we are every day made aware, is one of the earliest and most widespread affections to which civilized races are subject. Happily, it is one of those concerning the causes of which we have least occasion to doubt, and its prophylaxis, owing to its manifest direct relation to those causes, is more simple and obvious than that of any other ailment. It is largely a preventable disease, the existence of which among us on such a scale as statistics have revealed is a reproach to our boasted twentieth-century intelligence. The late Professor W. D. Miller's classical experiments on the fermentation of food-stuffs outside the mouth and its effect on teeth demonstrate clearly what is the main cause of dental caries; and there can be no excuse for us if we fail to adopt and recommend those measures that such experiments and clinical experience prove to be necessary for its prevention.

The systematic suppression of dental caries by the elimination of its causes would, I think, effect more for the general well-being of the nation than the aggregate brilliant surgical work that is being carried on in our general hospital wards and theatres devoted to operative

6 *Hern: Relation of Dentistry to other Branches of Medicine*

surgery, for it may be regarded as the ailment most inimical to the health of the body. It is obvious that the saving to the nation by its practical abolishment or great diminution would even pecuniarily be enormous. The frequency of its occurrence, so that it is popularly regarded as a sort of apanage of human existence, and the concealment within the mouth of its steady ravages, constitute it perhaps the most insidious of all the various forms of disease that sap vitality. Professor Osler reckons it as a factor in physical deterioration even worse than alcohol. One cannot be astonished at the frequent incapacitation for work among adult bread-winners, the rejection of multitudinous Army and Navy recruits, and the invaliding of thousands of soldiers in time of war simply on account of dental caries, when one considers that proneness to decay of the permanent teeth is such a common manifestation in early life. The adult, if so be he has come to value and to be careful of his teeth, cannot undo the mischief wrought on them in the days of his childhood and youth. Among the indirect causes of early decay may doubtless be reckoned the giving of food that is lacking both in quantity and quality: the initial dietary is such that the growing child's constitution is unfitted for the production of good permanent teeth, even though the condition of the milk-teeth should be unaffected.

It is a well-recognized fact that the health of women both before and after parturition has a potent influence on the condition of the teeth of their offspring. It is of prime importance, therefore, in order that the teeth of the young may be perfectly developed, to promote the well-being of mothers and expectant mothers by seeing to it that they not only suffer from no debilitating ailment, but also that they be properly nourished. In Blackburn a Nursing Mothers' Aid Society was lately established to encourage the breast-feeding of infants. To this end, arrangements were made to supply good plain and substantial well-cooked dinners to women of the poorer classes, both before and after their confinement, the stipulation being made that recipients of the meals should weekly bring their infants to be weighed. It would be well for the race if other towns would follow the excellent example of Blackburn.

It has been suggested by Dr. Sim Wallace, whether seriously or not I am unable to say, that before a woman is granted a certificate of marriage she should be required to pass an examination in hygiene and dietetics. Unhappily there is a growing tendency nowadays to regard artificial foods for infants as equal in nutritive value or even superior to mothers' milk. Possibly a wish to escape from one of the onerous

responsibilities of maternity is in some cases the father to this thought. Bottle-feeding, though it may have little or no effect upon the formation of the milk-teeth, is very frequently a poor preparation for the healthy development of the permanent set. A generally little-suspected but indubitably common contributor to the decay of both milk and permanent teeth is over-elaboration of the diet, more especially when the infant has come to acquire teeth. The food that is given to supplement or to replace the infant's milk-supply is of so soft and pulpy a character as to afford the newly arrived teeth hardly any employment; also, being specially tenacious and viscid, it occasions a long retention of fermentable particles on and between the teeth. The common result, as Dr. Sim Wallace remarks, is that the teeth get dirty and tender from want of use, later becoming carious.

The chemical properties of the food consumed are of no less importance in regard to the production of decay in teeth than are the physical. A diet consisting largely of starch or sugar, especially of the more easily fermentable varieties of sugar, as some of our members have demonstrated, is particularly prone to produce caries. In regard to these matters not merely the public but actually many persons engaged in the practice of medicine have much to learn.

But what is of paramount importance in the waging of war upon dental caries is the providing of information for children in the hygiene of the mouth. It is clear that the first necessity as regards school children is to secure that they shall receive systematic instruction in the care of the teeth. Further, the early manifestations of the disease must, as a matter of course, be adequately treated by stopping. It might with advantage be explained to children that those portions of the teeth that are exposed to friction, such as the cusps of the molars and the cutting edges of the incisors, are practically free from liability to decay, and that also those parts of the teeth that can be and are swept with the tooth-brush suffer comparatively little. Another exceedingly important matter is that friction is as necessary for the preservation of a healthy condition of the gums as it is for that of the teeth. It cannot, I think, be too strongly insisted on that the gums are firm and healthy just in proportion as they are subjected to friction, and that congestion and sponginess, the forerunners of pyorrhoea, are met with only in those situations where friction, natural or artificial, is least applied.

If instructions in accordance with these facts could be issued and practically enforced, a marked decrease in caries and oral sepsis would

8 *Hern: Relation of Dentistry to other Branches of Medicine*

be the speedy outcome, with concomitant untold effects for good on the physique of the nation. The gain in public health would ultimately prove to be far greater than could be attained by the appointment of a host of qualified practitioners to deal with dental disease already well established and actively progressing—disease that might have been prevented by the adoption of such simple precautions. It is futile to dream of combating the vast amount of dental disease existing in our midst by sending its myriad victims to our various hospitals and institutions that merely treat the effects of the disease.

It appears to me to be a question whether, as one of the foremost dental societies in the realm, this Odontological Section of the Royal Society of Medicine in one of its important rôles—viz., the prevention of disease—should not formulate and publish such instructions for the care of the mouth and teeth as are dictated by the latest scientific knowledge and clinical experience of its members, for I apprehend that the prevention of disease is a better and higher aim even than its cure, and it is as much a function of societies as of individuals.

I am fully aware that much more might well be said regarding the topics I have this evening touched upon, but I feel that I may wisely leave you to supply out of the stores of your own experience whatever you may find to be lacking in my discourse. It remains for me now to thank you most heartily for the kind attention you have bestowed upon what has necessarily been an all too perfunctory disquisition on matters of the greatest moment to our profession as well as to the public.

A Case of Infective Disease of the Jaws associated with the Absorption of the Teeth.

By A. HOPEWELL-SMITH, M.R.C.S., L.D.S.

THE ambiguity or indefiniteness of the wording of the title of the short paper herewith presented to this Section has been purposely designed to announce the circumstance that the writer is undecided as to the exact interpretation of the clinical and pathological conditions and the accurate appellation with which to label the cause or causes of the disease which is to be immediately considered. This uncertainty in his own mind has not been diminished, but rather increased, by discussing the supposed pathology with a few competent English authorities.

The case is possibly not quite unique; but, possessing very extraordinary characteristics, its ætiology is so difficult to determine, and the actual pathological phenomena which it exhibits so full of significance, that, as a result, it is raised to a high level of interest and importance. The history has been partly supplied by the dental surgeon who has been consulted by the patient and partly by the patient himself, and if it fails in many particulars to be of the utmost value, and lacks thorough completeness, it nevertheless throws some light on the question of radicular absorption and problems of diagnosis.

The patient, Mr. O., aged 44, has been under observation for a number of years, his mouth presenting very varied aspects as the disease advanced. Family history: The mother is edentulous: the father practically so. A sister, aged 25, has the right maxillary canine still unerupted, the cusp just appearing through the gum (October, 1909). There is no purulent effusion round the teeth. Past history: About fourteen years ago the patient, then being aged about 29-30, had a swelling under the mandibular first molar, which was very carious. Extraction under an anæsthetic failed to remove the tooth entirely. On examining the mouth at the time of operation, it was discovered that all the other molars—the upper and lower third molars not included—had their crowns only slightly erupting through the gum, although the premolars and front teeth were fully developed and occluded in a normal manner. As the conditions were extremely uncommon, and as the swelling “did not go down,” the patient was sent to the consulting surgeon to the

Dental Hospital of London, who, anxious to await further developments and give Nature an opportunity of correcting the abnormal state of the mouth and jaws, merely ordered an ointment for external use.

When the dental surgeon next saw the case an abscess had opened which would not heal. The patient was thereupon sent to the Dental Hospital, and the surgeon for the day endeavoured to remove the roots of the tooth, but failed. He was then seen by the dental surgeon of the Middlesex Hospital, and again operated upon, but with no greater success. Some long time afterwards the patient told his private practitioner that a piece of bone, or "something like it," came through the opening on the face, and then the abscess healed. All the partially-erupted molars except one have become shed spontaneously—i.e., got loose, and been removed from the mouth by means of the fingers, and are in a more or less similar condition, showing the signs of multiple absorption. The crown of a wisdom-tooth was, in December, 1908, felt with an explorer, buried in the jaw, on the upper right side.

The patient himself told the writer that, with jaws closed, the fingers could be inserted between the masticating surfaces of the molar teeth on both sides, showing that they never occluded in the normal manner, a space of one-sixteenth or one-thirty-second part of an inch intervening. For fifteen months there had been a certain amount of discharge of pus and swelling of the gum tissues. Trismus was present at times to a slight degree, and the patient also suffered greatly from insomnia. The maxillary right canine was sound, but had an abscess associated with it, and was therefore somewhat loose. Having meanwhile been crowned, it is now quite firm. Practically there has been no pain throughout the whole course of the disease, the discharge of pus from the back of the mouth being the most prominent symptom. There has been no tartar to speak of, and the general condition of the oral mucous membrane is, and has been, fairly good. The patient had rheumatic fever when he was aged 33, is somewhat delicate, and probably neurasthenic.

Present condition (July, 1909): The mouth is clean and well cared for. Tartar is absent round the necks of the remaining teeth. Great absorption of the alveolar processes has occurred. Upper jaw: Of all the maxillary teeth, the two canines, the second left premolar, and the root of the second right premolar remain, one of the former having been crowned. The latter, however, shows signs of periodontal disease, especially on its distal aspect, probably through loss of bone-substance of the jaw. Since the impressions were taken, June, 1909 (fig. 1), the

upper right wisdom-tooth has become shed. Lower jaw : Alveolar ridges are flat ; the teeth remaining *in situ* are the two lateral incisors, the canines, the two first premolars, and the left second premolar. The other teeth have been extracted from time to time, through loosening on account of the alveolar changes. Eruption of the right mandibular wisdom-tooth is now taking place. Unfortunately, the patient objects to having an X-ray photograph of this tooth taken *in situ*.

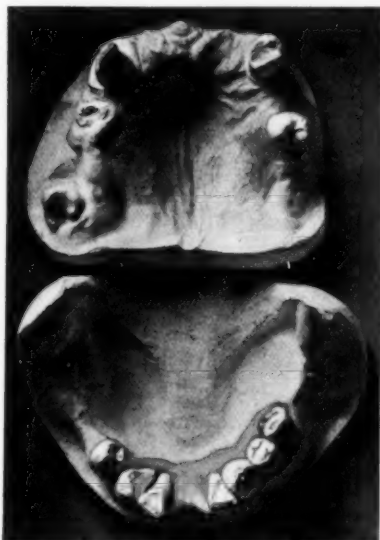


FIG. 1.

Plaster casts of the jaws showing the condition of the mouth in June, 1909.

Macroscopical Appearances.—Left mandibular second molar : On the occlusal surface of the crown there is a rounded cavity occupying the site of the postero-mesial cusp (hypoconulid), its greatest diameter being 4.5 mm. and shortest 3.5 mm. Extending downwards and backwards and towards the lingual side, it avoids the pulp chamber and opens externally below the cervical margin on the posterior surface by a small pin's-head point, on the lingual surface by two sinuous perforations (fig. 2), and in the radicular region in a large irregular shallow excavation, 9.5 mm. in length. The greatest area of absorption is found on the

mesial aspect (fig. 3). Here the enamel is unaffected, but immediately below, a large loss of dentine and cementum has taken place, measuring 12 mm. long by 8 mm. wide. The cementum is hyperplastic, the pulp chamber not exposed. The length of the tooth equals 21 mm. There is no transparency of the roots, which are confluent through the hyperplasia of the cementum. One minute apical foramen can be detected.

Right maxillary third molar: Crown is flattened antero-posteriorly. A large amalgam filling is situated on the buccal surface, and fills the



FIG. 2.



FIG. 3.

Fig. 2.—Left mandibular second molar: lingual aspect.

Fig. 3.—The same tooth: mesial aspect.

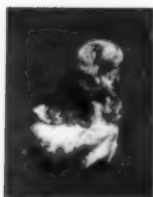


FIG. 4.

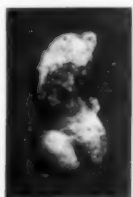


FIG. 5.

Fig. 4.—Right maxillary third molar: distal aspect.

Fig. 5.—The same tooth: buccal aspect.

site of the antero-external and antero-internal cusps. A discoloured patch of enamel is seen on the free edge. The mesial surface of the root is discoloured and presents three small hollows. A large area of absorption is observed on the distal and buccal surfaces (figs. 4 and 5), the former being entirely bored out except at the extreme apical region.

The roots are very hyperplastic and reflected backwards. The apical foramina are closed and invisible. The whole of the palatine root has disappeared, the consumed surface measuring 12 mm. by 6 mm. by 5 mm. The length of the tooth is 21 mm.

Right maxillary second molar: The crown is roughly quadrilateral in shape. There is a gold filling on the morsal surface at the part between the antero-external cusp and the ridge joining the antero-internal and postero-external cusps. The crown is otherwise free from caries. The buccal roots are fused together, rough, and greatly enlarged by hyperplastic cementum, the surface being very slightly attacked by the destructive process. The apical foramina are invisible. The palatine root is largely reduced in length by the rarefaction, slightly near the cervical region on the lingual aspect, more so at the apical portion. The length of the tooth to the apices of the buccal roots was 18 mm., the palatine root measuring 16 mm. (figs. 6 and 7).

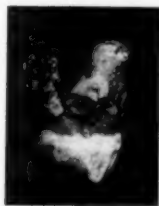


FIG. 6.



FIG. 7.

Fig. 6.—Right maxillary second molar: distal aspect.

Fig. 7.—The same tooth: lingual aspect.

Left maxillary second molar: The crown is non-carious. There is a tendency for it to become oblique in outline, its fissures and pits being pronounced. The enamel is entirely free from disease. The buccal roots are confluent, but discoloured. There are cavernous openings on all the surfaces, the greatest being over the innermost. It covers an area of 42 sq. mm. (fig. 9). The pulp is not penetrated. The palatine root, exceedingly honeycombed, is reduced in length. There are several excrescences of pathological cementum on the distal surface of the body of the tooth, at the junction of the roots (fig. 8). The length of the tooth, on its buccal aspect, is 20.5 mm., and 17.5 mm. on lingual side.

Left maxillary third molar: The crown here is non-carious, but flattened from before backwards. Enamel, while being discoloured, is

intact, except undergoing a small amount of loss on the mesio-lingual side. Two small deep pits are observed on the buccal aspect. All three roots are fused and thickened. The buccal roots on the cheek surface exhibit an absorption area 7 mm. and 4 mm. across the widest and narrowest diameters respectively, while distally, an irregularly formed cavity is seen, and mesially, two small depressions, closely situated. The palatine root displays the ravages of the disease best of all. On the mesial aspect the dentine and cementum, covering a superficies of 13 mm. by 7.5 mm., have been removed (fig. 11). The pulp is here apparently invaded. The length of the tooth averages 19.5 mm.

The edges of the excavations of all the teeth, on examination with a lens, are, generally speaking, rounded, everted, and smooth when dentine and cementum are destroyed; when enamel is involved, sharp and well defined. They nowhere exhibit the naked-eye appearances of having

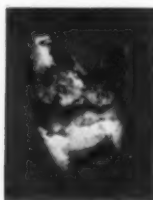


FIG. 8.

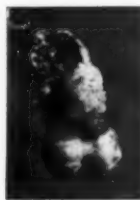


FIG. 9.

Fig. 8.—Left maxillary second molar: distal aspect.

Fig. 9.—The same tooth: buccal aspect.

been produced by osteoclasts, as Howship's foveolæ are probably entirely wanting.

The literature relating to absorption of the roots of the permanent teeth, it must be confessed, is somewhat disappointing. Tomes and Nowell [8] (p. 535) mention several cases, notably one, very briefly sketched, of a young person who had lost the six upper front teeth. "There was no assignable cause for this destruction." One vainly looked for some explanation as to the reason. Smale and Colyer [7] go further than the authors just named when they decide that the immediate cause of absorption of the permanent teeth is chronic inflammation of the dental periosteum, the inflammatory products, instead of organizing as in exostosis, producing absorption; it is therefore somewhat analogous to rarefied osteitis (p. 263, 1893 edition).

J. Sayre Marshall [6] divides the causes into predisposing and exciting. Of the former he mentions two classes, one of which is "dependent upon conditions associated with the tooth itself, and the other on conditions which are entirely foreign to the tooth." The causes of the first class are "death of the pulp, necrosis of portions of the pericementum [*sic*], and functional disease of the tooth due to loss of its antagonist." As exciting causes, "continued irritation of almost any form may inaugurate the resorptive process." Among the more common of the exciting causes may be mentioned chronic septic pericementitis, chronic dento-alveolar abscess, &c. In other cases, however, the resorption process seems to have no discoverable cause whatever. Again, Inglis [4], in the last edition of that excellent work, "A Text-book of Dental Pathology and Therapeutics," of which Henry Burchard was the originator, perhaps gets nearer to the real conception of the condition,



FIG. 10.

Fig. 10.—Left maxillary third molar: mesio-lingual aspect.

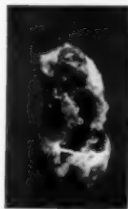


FIG. 11.

Fig. 11.—The same tooth: mesial aspect.

when he says (p. 575) "the proximate cause is probably in all cases a degree of irritation greater than that required to produce hypercementosis. Talbot's demonstrations of interstitial gingivitis, a term meant to include interstitial pericementitis, show that it is a frequent cause of both root and alveolar absorption." "The disease has been discussed by other writers as a result of rarefying pericementitis" (*sic*). According to this author, chronic apical abscess seems to be a frequent cause. Granulation tissue exists in the alveolar socket, being produced during "the periods of lessened pus formation." In conclusion, he says: "Some of the cases exhibit no tangible cause; the root resorbs apparently as the result of a peculiar reaction upon the part of the tissues of the individual, who may lose many teeth by this process—i.e., a

dyscrasia exists. The teeth may be non-carious and the pulps vital. In some of these cases neurasthenia, or a uric-acid diathesis, seems to have some association with the condition."

Problems of Diagnosis.—In reference to the present case several facts stand out strikingly. First, the molars erupted most imperfectly. Some unknown reason lies at the back of this. The ordinary physiological forces were not only retarded but in abeyance. They cannot have been affected by the deciduous dentition, as the permanent molars, of course, come up behind the milk teeth. There must be some constitutional disturbance at work, acting in an extraordinary manner, in producing this grave defect in the assumption of their normal position in the dental arch of the masticatory organs. The history of the right maxillary wisdom-tooth shows that its life duration only extended over about seven or eight months. There is complete absence of any of the usual causes of delayed or anomalous eruption of teeth, and its cause in the present case remains a mystery. Secondly, the molars were not necrosed—i.e., dead. In each, except one, the pulp was alive and protected by its wonderful physiological resistance from the attacks of the great pathological changes going on outside. Thirdly, the disease, whatever was its nature, was practically confined to the molar region. None of the other teeth were similarly affected. This is the most inexplicable part of the case. That the infection, if it was induced by pyogenic micro-organisms in the first place, should limit itself to the posterior parts of the oral cavity is truly surprising. Fourthly, the presence of pus in large quantities at times was a prominent sign and, no doubt, associated with the granulation tissue produced around the roots of the teeth. Fifthly, the molars, having shared a common affection which was spread over a considerable number of years, became shed spontaneously. Again one asks, "Why was this confined to the molar region?" "Why did not the teeth in the other parts of the mouth likewise suffer?" At the present moment the patient is erupting the last of the series—a right mandibular wisdom. Its fate will, no doubt, be similar to that of the others, which will prove that the pathological forces are still at work. Time alone will show. It is a misfortune that, as the patient objects to having an X-ray photograph taken, one cannot ascertain the exact conditions present to-day in the region of this tooth. It is also a misfortune that the teeth had not been preserved in a fresh condition on being shed.

Conclusion.—It is difficult, as has already been pointed out, to decide on the ætiology of this alveolar disease. With all diffidence the writer

puts forward his opinion as follows: There were two factors probably which played an important predisposing part in the production of the disease—viz., (1) the disuse of the teeth through lack of occlusion, and (2) constitutional debility. Periodontal disease, accompanied by the formation of pus, it was not in the ordinary acceptance of the term. The packing of food in the interdental spaces of the molars probably never existed. Neither can the symptoms be labelled those of diseases in which *pyorrhœa alveolaris* is found, for they are just the opposite of these. There is no granulation tissue in diseases which produce pyorrhœa; there is therefore no dental absorption. Pathologists [5, 6, 7, 8, 9] agree that absorption of bone is produced either by the agency of osteoclasts—lacunar absorption; by decalcification of the hard parts—halisteresis; by granulation tissue, through the round cells on its surface. Arguing by analogy, it is probable that the latter agency may be applied to this case, when the probable sequence of events would be, a bacterial invasion of the periodontal membranes of the functionless molars, setting up a chronic periostitis which resulted in organization and hyperplasia of the cementum. The bacterial influences not having been withdrawn, the chronic periostitis was further changed into granulation tissue which, occupying the sockets of the teeth, by means of small round cells possessing phagocytic properties, removed not only the alveolar bone but almost equally the dentine and cementum of the teeth.

The writer acknowledges with many thanks the deep debt of obligation he owes to Mr. Bower, of Luton, who has so kindly given him these unique specimens; and to Mr. Payne, of the Royal Dental Hospital of London, for photographing them for him.

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- [6] SAYRE MARSHALL. "Operative Dentistry," 3rd ed., 1909, p. 527.
- [7] SMALE and COLYER. "Diseases and Injuries of the Teeth," 1893, p. 263.
- [8] TOMES and NOWELL. "A System of Dental Surgery," 1906, p. 585.
- [9] TREVES. "A System of Surgery," 1895.

DISCUSSION.

The PRESIDENT (Mr. Hern) thought the case was unique. He had never seen a case exhibiting absorption of teeth to such an extent. He remembered one case in which a lady, a chronic sufferer from rheumatism, with rheumatoid arthritis of the knees and temporo-mandibular joints, had absorption of the roots of the upper maxillary canines. The teeth were very well developed and strong; there was no pyorrhœa. Her molars had the appearance, mentioned by Mr. Hopewell-Smith in his case, of being not fully erupted.

Mr. F. J. BENNETT thought the idea of a deficient blood-supply to the region might account somewhat for such forms of inflammation. The fact, as Mr. Hopewell-Smith pointed out, that the molar region was the one chiefly affected, the other regions being in good health, suggested that the non-eruption of the wisdom teeth and their functional inactivity, together with their early loss, might possibly be accounted for by some defects in the nourishing vessels.

Mr. H. BALDWIN said one knew well of the absorption which took place in roots and then in crowns of teeth which had been replanted or injured by a blow years previously. He wished to know whether Mr. Hopewell-Smith looked upon such a process as at all similar to those he had mentioned. The cavities in such teeth were filled with a highly vascular mass, which looked something like granulation tissue, which was active in absorbing teeth. The result appeared to him something like the result in Mr. Smith's cases, except that in his the absorption had followed a highly irregular and serpiginous course, the teeth appearing as though they were drilled through and through, as though worm-eaten, and most peculiar.

Mr. CLEMENT LUCAS observed that the only thing that struck him about the case was that the author said he was describing cases of non-erupted teeth or imperfectly erupted teeth. He would suggest that if those teeth were imperfectly erupted there must be some defective development at the site where the teeth should have come through at their proper time, and that in all probability as a consequence there was a lack of resistance to disease at that point, and therefore if an infective process were introduced it would be more likely to attack that part of the mouth than any other. That was the only thing that occurred to him in connexion with it. The absorption described by Mr. Hopewell-Smith was brought about, if he understood him correctly, by some irritating condition going on, and the speaker thought an infective process attacking the part which was badly developed had caused suppuration and granulating material, which was the secondary cause of the eating away of the roots of the teeth.

Mr. STANLEY MUMMERY asked whether Mr. Hopewell-Smith was able definitely to put syphilis out of the question.

The PRESIDENT said it seemed to him the condition was very different from the absorption that occurred in teeth that had been injured and replanted. There seemed to have been in the case under discussion some periostitis, showing increased deposition of cementum, and in other regions a kind of spongy absorption that had a quite uncommon appearance.

Mr. A. HOPEWELL-SMITH, in reply, said the explanation of the cause of the disease was extremely difficult to arrive at. He had asked several pathologists for their opinions, and they had all disagreed. It had been suggested that syphilis might account for it, but there was no definite history of that disease at all. One thought it was what Mr. Tomes had referred to as caries interna, and a third opinion was that it might be due to lack of development of the roots. A most eminent pathologist had told him that. But that was not so, because it was perfectly obvious from the measurements of the teeth that they were remarkably well developed. For the purpose of drilling holes to put the pins into the crowns of the teeth he obtained some special burs with round heads, and believed he broke half a dozen on the enamel in doing so because of its remarkable hardness. In the case mentioned by the President there was a history of rheumatism, and he believed that had a great deal to do with the case he had himself described. With regard to a deficient blood-supply accounting for the non-eruption of the teeth, mentioned by Mr. Clement Lucas, there was no evidence in the other parts of the mouth, or the face, or the jaws showing a deficient blood-supply; and, in addition, one would wonder why it should be confined to the molar region—which was the extraordinary part about it—and not attack the other portions of the mouth. With reference to the absorption being similar to that of replanted teeth or teeth that had suffered from rheumatism, as suggested by Mr. Baldwin, he did not think that was an explanation, because replanted teeth were dead teeth, and those under discussion were all alive except one. He thought Mr. Lucas had got as near to the truth as it was possible to get when he considered that the infective process, whatever it was, had attacked a part of the jaw which was fully supplied with blood. There might have been a lack of physiological resistance to disease in that hidden defect, but there was nothing external to show that there ever had been a developmental defect.

Papillomata of the Uvula.

By F. COLEMAN, M.R.C.S., L.D.S.

THE subject of this communication was a man aged 22. On the right side of his uvula, near its base, was a pedunculated growth, somewhat resembling a small raspberry, with a long, slender stalk $\frac{1}{4}$ in. to $\frac{1}{3}$ in. in length. The growth laid on the surface of the uvula, and its pedunculated nature was only detected when it was found that a probe could be passed freely underneath it; when lifted from the uvula it was seen to be attached by a fine, slender stalk. The patient was suffering from a chronic pharyngitis, so that his soft palate was almost completely insensitive, and even slight traction on the tumour scarcely produced retching. The patient was unaware of the papilloma, so that it had evidently caused him no inconvenience. The growth was very friable, and it was not until its whole substance was in the grasp of artery forceps that its attachment could be snapped across; this was done with curved scissors, removing with it a small button of mucous membrane at the base of its attachment. Its appearance gave one the impression that it would become rubbed off in the course of a few days, or weeks, and it was rather with the view of expediting matters that I snapped it off there and then, the patient having primarily attended the hospital for some other purpose.

Curiously, in the same week, I came across another similar case, also occurring in a man of about the same age, suffering from chronic pharyngitis. The papilloma, again, was attached to the base of the uvula by a long, slender stalk, which was only apparent on raising the tumour; its surface was covered with fine papillæ, again closely resembling, in shape and appearance, a small raspberry. I attended to his dental trouble, and then referred him to a surgeon with regard to his throat.

These cases impressed me with the fact that I had seen a good number of uvulæ in the course of my work, but yet could not recollect previously to have seen a similar condition. On making inquiries I could find no record of such cases in the usual text-books, nor was the condition mentioned in those special books on the nose and throat that I referred to. Tilley mentions that papillomata, amongst other forms of neoplasm, are recorded as taking their origin from the uvula. I could

find no reference to such cases in the *Index Medicus* for the years 1905, 1906, 1907 and 1908; however, in the *Surgeon-General's Index-Catalogue* I found two cases, recorded respectively by Dr. Freeman French¹ and Dr. Clinton Wagner.² These cases so closely resemble those I have already mentioned that a description of them is hardly needed; suffice it to say that the latter gentleman (Dr. Wagner) makes the following statement: "Failure to recognize the growth is chiefly owing to the fact that when the mouth is wide open, as during the examination for diagnosis, retraction of the soft palate, uvula and arches takes place, by which the growth is made to adhere closely to the surface from which its pedicle springs. The only way to avoid overlooking such growths is to attempt to move or dislodge with the probe any unusual prominence that may be seen at the places mentioned."

Dr. F. A. Bainbridge has kindly furnished me with a microscopical section of the growth, which shows it to be a simple papilloma. The macroscopical characters I have described can be seen on removing the slide.

Exostoses of the Mandible.

By F. COLEMAN, M.R.C.S., L.D.S.

NOTICING how frequently exostoses occur as palpable tumours on the inner aspect of the mandible, I was led to inquire into this question more fully and see whether one could not find some cause for this condition. For this purpose I made an examination of the greater part of the collection of skulls and jaws at the Royal College of Surgeons Museum; although this examination has thrown but little light on the cause of these exostoses, the research has enabled me to make a few observations as to the position and character of these osseous formations.

Exostoses of the mandible almost invariably occur on its inner aspect, and the positions they assume are fairly constant and characteristic. Previously, I had never fully appreciated the manner in which the lower molar teeth are, so to speak, slung in a bony hammock over the inner surface of the jaw, so that in many cases the third lower molar and, to a less extent, those in front are practically unsupported by the body of the jaw itself. (Hunterian Museum, Royal College of Surgeons,

¹ *New York Med. Record*, December 31, 1887.

² *New York Journ.*, February 25, 1893.

New Hebrides, 1153, Australian 1391 red.) This trough or shelf of bone carrying the molar teeth in many cases presents a well-marked ridge along its lower convex border, apparently serving as a buttress or stay to the overhanging inner alveolar plate, besides giving origin to the mylohyoid and superior constrictor muscles. This ridge commences behind and below the last permanent molar tooth and extends downwards and forwards as far as the second premolar tooth. The prominence of this ridge and the thickness of the plate of bone above it appear to bear some relation to the amount of lateral strain thrown on the lower jaw, as in thick-set jaws with firm, well-worn teeth, the ridge and the supporting bone are unduly developed, in some cases amounting to well-marked hyperostosis. (Royal College of Surgeons, Eskimos, 812 and 813.)

This condition of hyperostosis is almost invariably symmetrical and is especially well marked in the molar and premolar region, and absent in the incisor and canine region; in other words, is strictly limited to those teeth bearing a lateral strain during mastication. This hyperostosis frequently terminates in front in an abrupt margin, in some cases amounting to a definite exostosis. (Royal College of Surgeons, Peruvian, 1014/2.) Whether or not increased functional activity, associated as it usually is with increased blood-supply and hypertrophy, is the cause or a cause of this hyperostosis, I am unable to say, but the specimens I have brought forward seem to favour some such supposition.

Another variety of exostosis of the mandible occurs in the form of small, discrete, hemispherical elevations of dense, ivory-like bone situated almost invariably on the lingual aspect of the alveolus and frequently in the premolar region. These exostoses, again, are symmetrical even when multiple, and the teeth and alveoli in their neighbourhood are generally sound, so that they do not appear to have an inflammatory origin (*see* model and specimen). I do not volunteer to offer any reason for their existence; occasionally these discrete, hemispherical exostoses occur on other parts of the jaw, and, when unassociated with a tooth-socket, there does not appear to be the same disposition to symmetry. A third variety of exostosis occurring in the mandible presents itself as a large, nodular mass of dense ivory bone, generally growing from the region of the angle of the jaw. (Hunterian Museum, Royal College of Surgeons, 2209 C, and 2212.)

I am indebted to Dr. Keith, the Curator of the Hunterian Museum, for allowing me every facility for examining the skulls there placed, for giving me permission to show you some of these this evening, and for

referring me to a monograph by Bardeleben, of Jena, which appeared as a reprint from the *Transactions of the Berlin Anatomical Society*, April 22-25, 1908. The paper referred to deals with what is termed by the authors (Bardeleben and Hansen) the "torus mandibularis," and is based on an examination of about 400 skulls, chiefly from Scandinavia. The authors draw attention to the frequency of these tuberosities among the Esquimaux (80 per cent.) and their association with well-developed jaws and teeth, and believe that this may be a causal relation; on the other hand, they direct attention to the fact that these bony formations are also present in jaws where the teeth show but little attrition and in young skulls. From these observations the writers of the paper arrive at the conclusion that even if these formations may originally arise through mechanical causes, they tend to perpetuate themselves and finally become a racial trait, although a variable one.

From the point of view of diagnosis, exostoses do not offer much trouble. The discrete variety, occurring in association with a tooth-socket, and by far the commonest form met with, present as palpable swellings in the mouth, with the following characteristics: They form smooth, hard, sessile or hemispherical swellings; the mucous membrane appears pale and stretched, but is freely movable over their surface, and there is an absence of all inflammatory signs. These bony growths do not, as a rule, cause any inconvenience. The above characters, combined with a long history, when such can be obtained, and their tendency to occur symmetrically and in the premolar region, will confirm the diagnosis.

The largest exostosis of this nature that I have seen was brought to me by a surgeon for a diagnosis. The tumour occurred in a middle-aged man, and was about the size and shape of half a cob-nut, attached to the lingual surface of the mandible in the right premolar region; the tumour presented every characteristic of an exostosis, and a small palpable exostosis was present in a corresponding position on the opposite side of the jaw. This exostosis had never caused the patient any inconvenience, and except for its bulk the patient would not have been aware of its existence. We informed the patient that it was of no importance, but that, if he liked, he could see us again in six months. The man, however, lived in the north of England, so it is doubtful whether we shall ever see him again.

Treatment may likewise be dismissed in a few words. I have never yet known one of these symmetrical exostoses to require removal. The largest one in the Hunterian Museum (Peruvian skull, 1014/2) is only

about the size of half a cob-nut, and the presence of one that size in the case I have just recorded had given rise to no inconvenience.

In this connexion I thought it might be of interest to show another form of bony anomaly, this time occurring in the upper jaw under the name of "torus palatinus." As recently a full description of this condition has been given by Mr. Rickman Godlee, at the Surgical Section of this Society (February 9, 1909¹), I will limit my remarks to a brief description of my case. The patient in question, a woman, aged 28, presented a symmetrical, bony elevation, about the size and shape of an almond, in the centre of her hard palate, divided into two equal-sized lobes by a well-marked median groove. Her history was that the swelling had existed as long as she could remember, and thirteen years ago a mould was taken of her palate at the London Hospital. She was not aware of any other members of her family having a similar condition.

While at the Hunterian Museum I had an opportunity of seeing the unique collection of models duplicated from the specimens on which Mr. Godlee based his paper. I might incidentally mention that I could find no connexion of the condition of "torus palatinus" with that of exostosis of the mandible.

DISCUSSION.

The PRESIDENT (Mr. Hern) thought the cases communicated by Mr. Coleman were very interesting. Cases of exostoses of the mandible, however, were comparatively frequent about the position of the genial tubercles. The model shown of torus palatinus was quite a good specimen. Mr. Godlee, at the February meeting of the Surgical Section, read an interesting paper on the torus palatinus, and showed a large number of models and several skulls exhibiting the peculiarity in many shapes and sizes, from very small to quite large ones. He thought the Section would do well to procure models of as many specimens as possible of torus palatinus for the Museum.

Mr. CLEMENT LUCAS said he had been drawn to the meeting that evening by Mr. Coleman's communication. As he understood it, Mr. Coleman was inclined to explain the symmetrical exostoses as the result of Nature trying to support the pressure which was put upon that part of the alveolus during mastication. In that case it would correspond to what one was in the habit of seeing in cases of severe rickets associated with knock-knee, where a buttress was thrown out on the inner side, and often in severe cases the line of the interior border of the tibia became entirely obliterated and a new line was formed which ran

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Surg. Sect., p. 175).

up to the buttress. It seemed to him that the author's explanation was a very good one for a large number of cases, but he remembered a case of a gentleman who consulted him in reference to tuberous exostoses—not of the mandible, but of the superior maxilla—and in that case his explanation would be different from that which had been put forward by Mr. Coleman. He had treated the patient referred to many years before for syphilis, and when he came again complaining of these exostoses on his upper mandible—there were several hard lumps—he at the same time drew attention to two lumps on his head which were also giving him pain. Under anti-syphilitic treatment the tumours on the head diminished in size and the pain entirely disappeared; but he did not think, so far as his recollection went, that those exostoses on the superior maxilla disappeared, although they diminished in size, and in that way the patient was relieved. In one of the jaws that had been exhibited there was room for another explanation. He had noticed there was a supernumerary tooth forming a lump in one of the jaws. With regard to the torus palatinus, he should like to know whether in a certain number of cases the tumour might be due to an ossified node. It must be known to all members how frequently syphilis, both hereditary and acquired, attacked the hard palate, and many dental surgeons, he had no doubt, had aided surgeons in placing plates over those apertures which occurred, in his experience, more often in hereditary than in acquired syphilis, as a result of perforation. In nodes occurring on any bone, if the anti-syphilitic treatment was carried to some extent the symptoms subsided. If before treatment was adopted ossification of a node had taken place, it was exceedingly difficult to get absorption of that node afterwards. He wished to know whether in those cases in which a hard lump was found in the middle of the palate a certain number—he did not say all—might not be explained in that way, inasmuch as it was well known that a node in the middle of the hard palate in some cases caused thickening, in others perforation. He had seen them opened and the serum let out and bare bone exposed. It might be an explanation that dental surgeons who had had a large experience of the jaws would accept as explaining some of the cases of thickening in the centre of the palate.

Mr. F. J. BENNETT said, with regard to the buttress theory, he thought on consideration it would be hardly acceptable. Such exostoses did not occur at the period of highest functional activity of the masticatory apparatus, but occurred in the later stages of life and increased with old age. Therefore he thought it could not be put down to that cause at all. His own experience from several cases he had seen was that they always occurred in people of arthritic diathesis, either gouty or rheumatic, and it always seemed to him to depend on their constitutional conditions. With regard to the first specimen which was sent round, he would certainly not have called that an exostosis at all, or a buttress. If the jaw were looked at, it would be seen that the arch of the teeth was of very much smaller dimensions than the body of the lower jaw itself and the lower teeth were set in a smaller arch; therefore their roots appeared to be very much on the inner side of the jaw; the alveolus

naturally was round those roots and appeared as an overgrowth or outgrowth, instead of which the teeth were simply set a little more on the inner side of the jaw than usual. It was not at all an uncommon condition in well-developed jaws.

Mr. WARWICK JAMES said, with regard to the exostoses on the inner side of the mandible, he had been accustomed to explain them as an ossification occurring at the attachment of the mylohyoid muscle near its anterior end. If the mylohyoid ridge were followed along, it would be found that the point at which it became less marked in front corresponded with the situation of these exostoses. These bony projections are always symmetrical and are found in adult life; as Mr. Bennett had pointed out, they are more distinct as age advances, when the parts covering them are thinner. It is true that they are usually situated above the mylohyoid ridge, but this is accounted for by the direction in which the muscle fibres act—for instance, in swallowing. The pull may affect the periosteum and bone above the ridge, as the hyoid bone upon which they act is situated below and behind. Mr. Bland-Sutton has pointed out that an exostosis is often found at the point of attachment of a muscle, as in the case of the adductor magnus, the adductor longus, and other muscles. In this particular case the exostoses are often rounded, which may be considered an argument against this theory, as may also the situation above the attachment of the muscle, unless the explanation given above is accepted. In some of the jaws passed round the whole of the ridge and the bone above it showed very considerable thickening. In these cases the anterior ends would be very prominent, and markedly so when covered by the soft tissues.

Mr. H. BALDWIN said the question of exostoses on the alveolar processes was an interesting one, and he was glad the question had at last arisen before the Section. He had often wondered why somebody had not written a paper upon it. As a matter of fact, very little or nothing was known about the ætiology of these exostoses. Certain things that struck him forcibly about them were that when the exostoses were found on one jaw, they were almost always present on the other; that when present, they were always on the external surface of the upper alveolar process, and always on the internal surface of the lower alveolar process; that they were almost entirely confined to the canine and premolar region, sometimes extending to the molar region; and that they were distinctly not in the line of any muscular attachment. The upper ones were always perfectly free from the attachment of the buccinator and the lower ones were clearly free from the mylohyoid ridge. Both were often so pronounced that they grew outwards, and then turned upwards in the upper jaw and downwards in the lower jaw, so that they might be described as almost unciform, and it was very difficult to get a satisfactory impression of them. One could often hook one's finger-point right round them. Mr. Joseph Turner some time ago gave his opinion that such exostoses were due to the teeth near which they were situated being affected by pyorrhœa alveolaris. It had struck him (Mr. Baldwin) at the time that that was not necessarily so; and since

then he had examined quite a number of them to see whether there was always pyorrhœa in connexion with the teeth. The result was he had noticed pyorrhœa sometimes in connexion with the teeth in the neighbourhood, but certainly not always, and not even in the majority of cases. The teeth in the majority of cases in close proximity to the exostoses were perfectly free from pyorrhœa, and sometimes entirely free from any sign even of recession of the gum. He thought himself they were connected, as Mr. F. J. Bennett had said, with a rheumatoid or gouty diathesis, and he believed when the teeth became lost they in time disappeared by becoming absorbed. The theory of Mr. Coleman, that they were caused by the tendency of Nature to strengthen the bone on the weaker side of the alveolar process, was an ingenious one, but he did not think that theory would explain the facts. In the ordinary action of mastication the lower premolars and molars tended to be thrown outwards against the external alveolar plate, and the upper corresponding teeth tended to be thrown inwards against the internal alveolar plate, and that was the opposite side to that on which the exostoses were found.

The PRESIDENT said, in regard to the multiple exostoses which were found sometimes right along and above the alveolar edge on the buccal side of the upper jaw, he rather agreed with Mr. J. G. Turner that they were due to chronic congestion of the periosteum associated frequently with pyorrhœa; but with regard to the symmetrical exostoses that occurred in the lower jaws, they were usually situated far below any congestion which might occur from pyorrhœic conditions. He had been making a little museum of specimens of that variety of exostoses for some time, and had found they varied greatly in size and shape; in one of those in his collection the exostoses formed symmetrical, spur-like projections that were $\frac{1}{2}$ in. long, and he had had to put in a plate in this case, and was obliged to cut it away to arch over the projections. He suggested that anyone who had such cases should take models and present them to the Museum in order that more might be learnt about the position and frequency of these exostoses than was known at present. With regard to the torus palatinus, he could not agree with Mr. Clement Lucas that it was in any sense connected with syphilis. He had handed Mr. Godlee about five models of torus, and in none of these cases was there the remotest suspicion of any specific cause. Some he had watched for a considerable time, and many of them had histories extending over a very long period. He should be very happy to present to the Society duplicates of the models of the five cases he gave to Mr. Godlee for his communication. Curiously enough, he had that day seen a patient with a torus palatinus that had been present for probably twenty years, according to the patient's account.

Mr. F. COLEMAN, in reply to the President, said the third variety of exostoses referred to in his paper presented itself as a large mass of dense ivory bone in the region of the angle. He had not laid much stress upon that last variety because it was not directly connected with the communication he had brought forward, but only put in to make the subject more complete. With regard to

Mr. Lucas's remarks concerning the thickening of the inner alveolar plate forming a sort of buttress comparable to the buttress seen in rickety cases, it was that likeness which made him (Mr. Coleman) consider whether it might not have a similar function in strengthening or supporting a part which was subject to a good deal of pressure. Mr. Coleman did not think, however, that any of the cases presented the rickety element. There was certainly no history of syphilis in the torus palatinus case, and personally he should have thought it a very unusual place to get a syphilitic bony node. Mr. James had thought the condition of hyperostosis might be due to an ossification of the mylohyoid muscle, but Mr. Coleman said there were a few rather strong reasons against that view. First of all, it would not be the kind of muscle that would ossify, because it had not a tendinous attachment to the jaw; it was generally a muscle ending in a tendon that ossified. Then, again, the exostoses did not seem to be situated at a place where the mylohyoid was attached, and they also occurred in early specimens.

Odontological Section.

November 22, 1909.

Mr. WILLIAM HERN, President of the Section, in the Chair.

Some Observations on the Bacteriology of Pyorrhœa Alveolaris, and the Treatment of the Disease by Bacterial Vaccines.

By J. W. EYRE, M.D., and J. LEWIN PAYNE, M.R.C.S., L.D.S.

PYORRHEA alveolaris, caries alveolaris specifica, alveolar osteitis, chronic suppurative periodontitis, periostitis alveolo-dentalis, or Rigg's disease, may be defined as a chronic inflammation of the gum margins and periodontal membrane, resulting in absorption of the surrounding alveolus. It is characterized by a persistent purulent discharge (which can be made to well up round the teeth by pressure over the sockets), and results in the loosening of the teeth situated in the affected area, and the formation of masses of granulation tissue in the interdental spaces; and, finally, if unchecked, causes the loss of the teeth, which one by one fall out. In advanced stages of the disease constitutional symptoms are the rule, and include muscular and joint pains (generally referred to by the patient as rheumatic), persistent headache, intestinal stasis, flatulence and indigestion; general malaise and anæmia, chronic pharyngitis and laryngitis; acute, subacute, or chronic cervical adenitis, acneiform and pustular skin eruptions, particularly of the face. Occasionally the anæmia is of a very profound type, and may be accompanied by symptoms of severe gastro-enteritis and toxæmia; and, rarely, a sudden exacerbation may terminate in ulcerative stomatitis, septic broncho-pneumonia, septicæmia, and death.

From the point of view of the bacteriologist, the presence of pus is the important feature in connexion with this disease, for at first

sight it would appear that the examination of this material would at once afford valuable information concerning the precise ætiology of the disease, and without such knowledge treatment is necessarily unscientific. Many observers have applied themselves to the study of pyorrhœa alveolaris (and their work will be found summarized in an Erasmus Wilson lecture delivered before the Royal College of Surgeons early in 1907 by Goadby¹), but the results obtained have been far from commensurate with the labour expended. The reason is not far to seek. The pus obtained from the sockets of the affected teeth in all these cases is teeming with representatives of an almost incredible number of different species of bacteria; and, further, so imperfect are our methods, even in the present year of grace, that it is impossible to persuade a tithe of these different species to propagate themselves upon nutrient media under the artificial conditions of the laboratory. Other species, again, grow within the culture tubes for two or three generations, but, being unable to adapt themselves to a saprophytic existence, die out before any detailed study of their biological characters can be completed. Some of the various micro-organisms present in the pus, however, belong to well-recognized types of pathogenetic and pyogenetic bacteria; others, again, are hardy saprophytes derived from food and air; these can be readily induced to grow upon laboratory media, and can be easily identified; but bacteria belonging to these classes form only a small proportion of the inhabitants of pyorrhœal pus, and we find that the majority of those investigators who have studied the disease have hesitated to commit themselves, and have referred to the bacteria isolated as "short, thick bacilli," "staphylococci forming yellow colonies," &c., so that it is difficult for their successors to correlate the observed facts.

Simms,² working at the subject in my laboratory some four years ago, confined himself to the study of ten cases of pyorrhœa, and from them isolated *Staphylococcus albus* and *aureus*, *Streptococcus brevis* and *longus*, *Bacillus maximus buccalis*, and *Bacillus fusiformis*, in addition to four other types of staphylococci and four more types of bacilli which did not conform with any degree of exactitude to any, at that time, recognized organisms. In these ten cases *Staphylococcus aureus* occurred twice, *Staphylococcus albus* once, *Streptococcus pyogenes*

¹ "Pyorrhœa Alveolaris," *Lancet*, 1907, i, p. 633.

² "Observations on the Bacteriology of Pyorrhœa Alveolaris," *Trans. Odont. Soc. Gt. Brit.*, 1907, xxxix, p. 164. "Pyorrhœa Alveolaris: its Bacteriology in Relation to Treatment," *Trans. Manch. Odont. Soc.*, 1908.

longus once, and a coccus closely resembling the *Micrococcus catarrhalis* ten times. In addition *Streptococcus brevis*, *Spirilla* and *Bacillus fusiformis* occurred in every case. Goadby, two years ago, in the lecture above referred to, records his observations in ninety cases of pyorrhœa. He was unable to determine that any one micro-organism occurred with sufficient regularity and constancy to warrant the opinion that it was casually associated with the disease, and he apparently inclined to the opinion that the pus formation indicated an infection with pyogenic bacteria, secondary to an infection by some at present unidentified organism, and evidently regarded the stomatitis and gingivitis of early life as corresponding to the primary infection, which in the adult comes under observation only in its later stages, and when a secondary pyogenic infection is well established. He mentions having isolated *Staphylococcus aureus* and *albus*, together with representatives of groups whose members, while closely conforming to these well-known species, vary from them in some minor characteristics; he mentions also a group of Gram-negative cocci whose cultural reactions do not conform exactly to any recognized type. He further states that of the streptococci he had isolated, two were probably related to the so-called *Streptococcus faecalis*, but that all were of the "brevis" type. He also isolated numerous bacilli, but mentions by name only *Bacillus fusiformis*, grouping the remainder under such characters as lactose fermenters, diphtheroid bacilli, and so on, and detailing their chief cultural characters; but attempting no further identification. Indeed, no further identification is possible without confusing the ultimate issue by labelling as distinct types organisms which, in the near future, will prove to be merely varieties of other and better-known species; for at the present moment our knowledge of the effect of environment upon the biochemical characters of bacteria is in an extremely nebulous condition, and there is no doubt that some, at any rate, of the organisms recorded in recent years as occurring in the mouth only are merely familiar saprophytes, hiding their typical characters under more or less unimportant and transient attributes—attributes which have been called into prominence by the peculiar environmental conditions under which they maintain the struggle for existence.

Again, last year, Goadby¹ recorded two further cases in which he regarded *Staphylococcus aureus* and *Staphylococcus albus* respectively

¹ *Brit. Med. Journ.*, 1908, ii, p. 477.

as the infecting organisms. Many of the bacteria isolated by Simms and Goadby were pathogenic for such laboratory animals as the rabbit, guinea-pig, and mouse, but attempts to reproduce the disease in the form it assumes in man have hitherto failed. The toxæmic symptoms have, it is true, been reproduced by Goadby as a result of the inoculation of the pus obtained from pyorrhœa cases into guinea-pigs; but pure cultures of isolated bacteria have failed to produce similar results.

Hence it will be seen that no one or more organisms hitherto detected in the pus can be regarded as "specific," when judged by the classical criteria of Koch as promulgated towards the end of the last century. We may remind you that these criteria are:—

- (1) That the organism shall constantly be present in the lesions of the disease under investigation.
- (2) That the organism shall be capable of isolation and cultivation outside the animal tissues.
- (3) That such cultivations, when injected into suitable animals, shall be capable of reproducing the original disease.
- (4) That the organism shall be again recoverable from the lesions of the artificially induced disease.

Nowadays, however, the results of Sir Almroth Wright's striking researches into immunity have provided us with further means for determining whether or no any given organism plays an active rôle in the pathological condition with which it is associated. Sir Almroth Wright, I may remind you, has demonstrated the presence in the blood-serum of bacterial antibodies which he has named opsonins. The function of opsonin is to so alter the bacteria invading the tissue that they are readily ingested by the leucocytes attracted to the site of infection. We do not propose to discuss the various opinions regarding the nature and the source of this substance. Suffice it to say that although perhaps it is not possible to measure the amount of opsonin present in an individual's serum with mathematical exactitude, measurements can be obtained which enable us to compare the bacteriotropic power of the patient's blood from time to time. These measurements, opsonic indices as they are termed, are expressed in percentages of unity, and in the normal individual variations from unity are found to occur in the amount of opsonin for any given organism between the extremes of about 0·8 and 1·2, an index below or above these extremes being held to indicate infection with the organism in question.

This forms one of the means at our disposal. Another is afforded by the result of treatment. Having determined, from the laboratory

point of view, that the patient is infected with a given organism, an attempt is made to immunize the individual by injecting killed cultures of that organism into the patient in such a manner as to increase the amount of available antibody. If the organism employed in the injections is actually the one responsible for the pathological condition, an excess of opsonin will be elaborated and will lead to an improvement in the clinical condition of the patient. Such improvement following the inoculation of suitable vaccine is the second of the means that Wright's work has placed at our disposal.

Goadby applied the first of these two tests to the micro-organisms he isolated from thirty-six of his cases of pyorrhœa, and was able to satisfy himself that micrococci of various groups were the responsible organisms for the various cases; next by the administration of vaccines prepared sometimes from one organism, sometimes from a mixture of several bacteria, he was able to so stimulate the production of antibodies as to induce a return of the diseased mouth to a normal condition in a large number of his patients.

Now, during the progress of the investigations carried out by Simms, our attention was drawn to the fact that in the specimens of pus derived from different patients, or from the same patient at different times, the organisms constantly present were the cocci, spirilla, and fusiform bacilli, and of these only the cocci consistently appeared in the cultivations established from the pus. Again, of those cocci which could be isolated, four species could be readily identified—viz., *Streptococcus brevis*, *Streptococcus pyogenes longus*, *Streptococcus lanceolatus*, one which was regarded as *Micrococcus catarrhalis*, *Staphylococcus aureus*, and *Staphylococcus albus*. The first of these was present in every culture, but as the work of Washbourn¹ and Goadby—communicated to the Odontological Society in 1896—had already conclusively proved this streptococcus to be a harmless saprophyte, we considered that it could at once be disregarded; the five remaining cocci are all well-known pathogenic microbes possessing marked pyogenetic properties, and it seemed possible, if not probable, that one or more of them were responsible for the suppuration, and that the other forms of bacteria, often weird and artistic in shape and contour, probably led a saprophytic existence in the pus and merely served as will-o'-the-wisps to entice observers from the real track into intricate and confusing side paths which end in a morass of confusion. And we may at once define our

¹ "Some points in connexion with the Bacteria of the Mouth," *Trans. Odont. Soc.*, 1896.

attitude by saying that we do not regard pyorrhœa as a specific disease in the sense that it is due to infection by one particular and specific micro-organism. On the contrary, we regard the condition as a suppuration due to one or other—or more than one—of the ordinary pyogenetic bacteria, chiefly pyogenetic micrococci, which owes its peculiar characters and prolonged course simply and solely to the accident of its anatomical position. Our observations all point to the sapræmic and toxæmic symptoms (which, after all, are the ordinary symptoms associated with prolonged suppuration wherever situated) being due to the absorption of septic material from the alimentary tract—i.e., due to the constant swallowing of small quantities of fœtid pus.

Until recently we were unable to go into the matter more fully, but as soon as the vaccine out-patient department at Guy's had been established and properly organized, at the request of our colleagues in the dental department we devoted some attention to the subject, with the result that the present series of cases gradually accumulated. In this series any bacilli observed in the pus were totally disregarded—even when, as sometimes happened, they appeared also in the culture tubes—and our attention has been restricted solely to the micrococci that developed upon the nutrient media which had been sown with the pus from tooth-sockets; and this line of investigation has, we are inclined to believe, been attended with a larger measure of success when applied to the treatment of the disease than can be accounted for by mere coincidence. Before, however, proceeding to discuss the bacteriology of the cases which form the basis of the present communication, a few words are necessary with regard to the methods employed.

Collection of Pus.—In collecting the pus the patient was instructed to grasp the lip opposite the affected teeth with the forefinger and thumb of each hand and draw it away from the gums; in some cases a small roll of absorbent wool was packed into the sulcus between the alveolus and the lip. The gum margin was next wiped with a sterile swab of cotton wool mounted on the end of a stick, and the gum itself dried with a second sterile swab. Then with another swab firm pressure was made on the gum over the root of the tooth. The first drop or two of pus that exuded was mopped up with a third sterile swab and the pressure continued, and the pus that next exuded was collected on still another sterile swab, or by means of a stout platinum needle. This pus was employed for the purpose of making coverslip films. Finally, more pus was expressed in a similar way and used to inoculate tubes of nutrient media.

Microscopical Examination of Pus.—At first McConkey's capsule stain was used to stain the films of pus after preliminary drying and fixation by heat. In the later work this stain was discarded, and preference given to Leishman's modification of Romanowsky's stain, thus dispensing with the fixation of heat, as fixation is affected by the methyl-alcohol in which the stain is dissolved. As, however, the film is frequently stained rather faintly, we are in the habit of further treating the film with a 0.1 per cent. aqueous solution of methylene blue. This has the effect of intensifying the blue stain already taken up by the bacteria without altering the magenta colour of the cell nuclei, or the red colour of the blood-disks. A second film was always stained by Gram's method. The microscopical examination of these films was, of course, carried out with a $\frac{1}{12}$ in. immersion lens. The appearances presented by pyorrhœal pus from the various cases were very similar. In addition to the pus cells, if, as so frequently happens, there had been oozing of blood from the gums during the pressure, a number of red blood-cells and leucocytes were seen. Of these latter, eosinophile cells certainly predominated. The bacteria present could be roughly divided into bacilli and cocci. Of the bacilli, *Bacillus fusiformis* (presenting many variations in size and shape) was constantly present, also numerous spiral rods, varying in thickness and in the intensity of their staining, and numerous other filamentous forms, together, usually, with some type of *Bacillus xerosis*. Many other varieties of bacilli were also present, varying widely in shape and size. Numerous micrococci were also seen varying in size and arrangement as well as in intensity of staining, usually including staphylococci, streptococci, diplococci, and tetrads. The films stained by "Gram" assisted in differentiating these micrococci, as some of the staphylococci, diplococci, and tetrads retained the stain in this method, while others did not. The streptococci invariably stained by Gram's method.

Cultivations.—The medium we have selected for the preliminary cultivation of these organisms has almost always been blood agar; that is, standardized nutrient agar to which sterile citrated blood—human or rabbit—has been added whilst the medium was in a fluid condition. The agar was then allowed to set with the tube at a slight angle from the horizontal, so as to get a long sloped surface. By inoculating several tubes in series it was often possible to obtain pure cultures of various organisms from the later tubes of each series. Whenever it was necessary to plate out the organisms in order to isolate them, one or more of three media were employed—standardized agar, nutrose litmus agar, or blood agar.

IDENTIFICATION OF THE RESPONSIBLE ORGANS.

After isolation in pure growth of the various micrococci present in the original culture, the patient's blood has been tested against each of the micrococci in turn, in order to determine the amount of opsonin present for each. That organism, or organisms, to which the index has been particularly low—say 0·5 or lower—or particularly high—say 1·3 or higher—we have regarded as responsible for the infection. While the opsonic estimations were being carried out the organisms themselves were being subcultivated and identified, and so, as soon as the organism we had determined upon as the result of the blood-test had been identified as corresponding to one or other of our "stock" laboratory cultures, the index of the patient's blood was tested against this stock-"known" organism. If concordant results were obtained the preparation of a vaccine for the treatment of the patient was proceeded with. This is the procedure we have followed in determining the responsible organism in the series of cases we are now discussing, and the results may be tabulated thus:—

ORGANISMS ISOLATED FROM PUS OF PYORRHEA CASES AND REGARDED AS THE RESPONSIBLE BACTERIA.

<i>Micrococcus</i> (staphylococcus) <i>pyogenes aureus</i>	2
<i>Micrococcus catarrhalis</i>	9
<i>Micrococcus catarrhalis</i> and <i>Streptococcus pyogenes longus</i>	11
<i>Streptococcus pyogenes longus</i>	7
<i>Streptococcus lanceolatus pneumoniae</i> (pneumococcus)	4

VACCINES.

Whilst dealing with methods, the procedure adopted in the preparation of the vaccines which were subsequently used in so many of the cases may be mentioned. The culture intended for the preparation of a vaccine was always made upon nutrient agar (except in the case of the pneumococcus, which was, of course, invariably planted upon blood agar) and incubated for twenty-four hours at 37° C. After examination of the growth to determine its purity, it was emulsified in the tube in which it was originally grown with some 5 c.c. of 0·1 per cent. saline solution. It was then transferred to a sterile test-tube, already provided with sterile glass beads, and placed in an electrical shaker for ten minutes. The enumeration of the organisms present in the emulsion was carried out by means of Wright's method, in which equal volumes of

the emulsion and normal human blood are mixed, spread as a blood film, stained, and the ratio between red cells and bacteria worked out. The emulsion was then killed by exposure to a suitable temperature in a water bath and a portion of it diluted with 0.25 per cent. tricresol solution until each cubic centimetre contained 100 millions of bacteria. Further dilutions were then made and separate doses corresponding to 5, 10, 50, or 100 million, &c., were put up, each in a small sterile glass bulb and sealed ready for use.

SELECTION OF CASES FOR OBSERVATION.

Of our series of thirty-three cases of pyorrhœa alveolaris bacteriologically examined, twenty-six have been systematically treated by the administration of suitable vaccines; two, by failure to attend, may be said to have refused treatment; whilst five have only come under observation during the past four weeks. Here we must point out that these cases have not constituted random samplings from those attending the out-patient department of our hospital, nor are they consecutive cases, but every one of these cases has been specially selected with reference to one or more of the following points: (a) Acuteness and intensity of the suppurative process; (b) extent of the disease and number of teeth involved; (c) intractable character of the disease and resistance to all ordinary methods of treatment.

The few cases seen in private may also be regarded as having been specially selected under one or both of the headings *b* and *c*, for treatment has been undertaken only at the request of the medical man in charge and with the permission of the dental surgeon, as a last hope, and when it has appeared that most of the teeth would have to be sacrificed.

Disregarding the two untreated cases, the five cases in which treatment has been followed for less than one month (although in three of these improvement is already marked), and also the case that died just after treatment was instituted, we have two cases which were diagnosed in the laboratory as due to *Micrococcus pyogenes aureus*; six to *Micrococcus catarrhalis*; six to *Streptococcus pyogenes longus*; and three to *Streptococcus lanceolatus pneumoniae*. Eight other cases were regarded as due to double infection with *Micrococcus catarrhalis* and *Streptococcus pyogenes longus*. The accuracy of the inferences we have drawn from the results of our laboratory experiments has, we consider, been amply confirmed by the results of the treatment; for the

treatment in each of these cases has been planned and carried out in accordance with the laboratory findings.

TREATMENT.

This was, primarily, by means of vaccines. As a preliminary the mouth was mechanically cleansed—a procedure which frequently extended over two sittings—the teeth were scaled, and the pockets washed out with an antiseptic, such as formaldehyde 10 per cent., peroxide of hydrogen, tincture of iodine, copper sulphate, &c. These necessary measures occupied the interval between taking the cultivations and the first injection of vaccine.

An interesting point in connexion with the cleanliness of the mouth is that no fewer than five of these twenty-five patients were members of the Jewish race (four females and one male); the exposed surfaces of the teeth in each instance were in excellent condition and were remarkably free from tartar, so that the amount of scaling to be done was quite trivial, yet in each case the infection was extensive and severe.

When vaccine treatment had been commenced a mouth wash was prescribed, either of formaldehyde 0·2 per cent., or peroxide of hydrogen 5-10 vols. This was given rather with the idea of directing attention to the condition of the mouth, and to impress upon the patient the desirability of washing away the excess of pus; for, needless to remark, no bactericidal action of any great value is to be expected from such feeble antiseptics during the limited period they would remain in contact with the infected gums. In addition to these measures and attention to the general health, vaccines prepared from the organism actually obtained from the pus in each case—that is to say, “autogenous vaccines”—were systematically administered by subcutaneous injection into the abdominal parietes; the size of the dose, and the intervals between the doses being controlled, in the earlier cases, by the movements of the index, which represented the patient's resistance to the organism employed in the vaccine; and in the later cases by direct clinical observation of the affected tissues. Rapid improvement was noted in most of the cases; some, however, were distinctly more resistant to treatment. The general impression we have derived from our observations is that the success of the treatment is influenced by the ætiological factor—in other words, the responsible bacterium—in each case. Pyorrhœa due to *Micrococcus*

pyogenes aureus yields most readily to treatment; next, that due to the pneumococcus; then comes that due to *Streptococcus pyogenes longus*. More troublesome is the pyorrhoea due to *Micrococcus catarrhalis*, and most intractable of all that due to a double infection with *Micrococcus catarrhalis* and *Streptococcus longus*.

Many of our cases we record as "cured," and it will perhaps be well if we state precisely what we wish to convey by the use of this term. Patients with pyorrhoea alveolaris well advanced when they first come under observation usually volunteer the information that the mouth feels uncomfortable because mastication is positively painful, owing to the fact that the teeth are loose. Many, in addition, complain of general ill-health, and in particular of arthritic (rheumatic) pains. Examination shows further the presence of pus in the tooth-sockets, absorption of the alveolus, and recession of the gums. The reverse of all these symptoms and signs—except the two last—we hold to constitute cure; that is to say, if the affected teeth are firm so that mastication is painless and the mouth comfortable, no pus can be expressed from the sockets, the pains and other symptoms of ill-health have disappeared, and the patient feels "quite well," then we are satisfied that the patient is cured. The recession of the gums, if at all marked, is associated with loss of bone—a condition which no amount of treatment of any kind is able to remedy. The clinical details of some typical cases may be summarized as follows:—

(I) RESPONSIBLE ORGANISM—MICROCOCCUS PYOGENES AUREUS.

(1) A. B., female, aged 23. April, 1908, to June, 1908: All teeth loose; mouth to be cleared for complete upper and lower dentures. Complaints of rheumatism past three or four years, and indigestion. Five doses ranging from 50 millions to 100 millions of appropriate cocci injected at average interval of fourteen days; duration of treatment, seventy days; cure extends over seventeen months. Present condition: No pus; gums healthy, two central upper incisors extracted; no rheumatism, digestion perfect.

(2) E. M., male, aged 37. September, 1908, to November, 1908: All teeth loose; all four canines and two lower central incisors extracted, remainder as far outwards as the upper and lower molars to follow; nine doses of from 50 millions to 250 millions injected at average intervals of seven days; duration of treatment, seventy days; went to Africa, last heard of "quite well"; threatened teeth still retained; duration of cure, nine months (probably twelve months).

(II) RESPONSIBLE MICRO-ORGANISM—STREPTOCOCCUS LANCEOLATUS
PNEUMONIÆ.

(3) E. W., female, aged 39. July, 1908, to December, 1908: Teeth gradually getting loose since 1906; gums swollen, spongy, and of dark colour, pus oozed from socket of almost every tooth, brown rings of hard tartar at necks of teeth and softer tartar over crowns; twelve loose teeth extracted before vaccine treatment commenced. General symptoms: Headache and general lassitude, pain in knee-joints. November 10, 1909: General health much improved, headaches gone; there is no pyorrhœa, the gums are healthy, teeth quite firm, but need scaling.

(4) R. C., male, aged 52: Many teeth loose, considerable amount of pus, patient seriously ill (? malignant growth); two doses (5 millions and 10 millions) at interval of six days; died a week or so after second injection; no post-mortem permitted.

(5) B. C., female, aged 45. May, 1908, to July, 1908: Mouth very foul, all teeth loose, profuse suppuration, rheumatism; six doses (10 millions to 100 millions) injected at intervals of nine days; duration of treatment two months, duration of cure sixteen months. Present condition: No pus, gums healthy, no rheumatism.

(6) L. C., male, aged 19. March, 1909, to May, 1909: Many teeth loose, considerable pus, arthritic pains; twelve doses (5 millions to 50 millions) at six-day intervals; duration of treatment eighty days, duration of cure six months. Present condition: Mouth normal, no teeth lost.

(III) RESPONSIBLE MICRO-ORGANISM—MICROCOCCUS CATARRHALIS.

(7) R. M., male, aged 39, checker at Smithfield Market. February, 1908, to May, 1908: Teeth loose for several months, mastication difficult, gums a deep purplish colour, bled readily on pressure, and pus oozed from most of the sockets, occasional gastro-enteritis, and pain in shoulder-joint for several years; discharged cured. November 10, 1909: The gums generally are not unhealthy, but a little pus can be obtained on pressure around $\frac{4}{1} \frac{1}{1.5}$; the arthritic pains have disappeared, and he can masticate without discomfort.

(8) J. C., male, aged 44, carpenter's labourer. March, 1908, to May, 1908: Pain, looseness of teeth, inability to masticate food; gum and alveolus had receded from nearly all teeth, and pus obtained on pressure at gum margin; black rings of tartar beneath gingival margin. Discharged cured.

(10) W. S., male, aged 43, butcher. May, 1908, to July, 1908: Teeth loose and painful for several months, latterly great difficulty in masticating food; gums red, swollen, and painful to pressure, pus could be expressed from all

teeth in front of the second premolars; all the incisors were quite loose. Discharged cured. November, 1909: The upper and lower incisors a little loose, but there is no pus nor tenderness, and power of mastication is quite good; no teeth were extracted.

(IV) RESPONSIBLE MICRO-ORGANISM—STREPTOCOCCUS PYOGENES LONGUS.

(16) R. G. L., male, aged 60, painter. February, 1908: Trouble commenced in May, 1906; pus obtained from socket of every tooth, gums turgid and purplish, tartar; arthritic pains for several years and mental depression. Nine hopelessly loose teeth extracted. November, 1909: Gums and teeth in mandible quite firm and healthy, ^{5,4,3} are loose, but there is no sign of pus. Feels better than for several years past, and there is no arthritic pain.

(19) S. J., female, aged 35, married. November, 1908, to January, 1909: Dull, aching pain and swelling of jaws, gums loose and spongy, several hyperæmic patches about mouth; every tooth affected, pus oozed from sockets, breath foul; all lower teeth except the second premolars extracted ten months before commencing vaccine treatment; headaches, fatigue, and lassitude. November, 1909: Lower denture worn and mastication restored, pyorrhœa cured, general condition improved.

(V) RESPONSIBLE MICRO-ORGANISMS—MICROCOCCUS CATARRHALIS AND STREPTOCOCCUS PYOGENES LONGUS.

(24) M. M., male, aged 44, clerk. October, 1908, to November, 1908: Treated for pyorrhœa alveolaris by local methods, three or four roots extracted; the suppuration being still well marked, vaccine treatment advised. Mouth well cared for, practically no tartar; gums looked clean but anæmic. A considerable quantity of pus could be squeezed from the sockets of ^{1,2,3} and a lesser amount from ^{4,8} _{7,3,4,5}. Flatulence, constipation, headaches, occasional joint pains. November, 1909: ^{1,2,3} quite firm and painless; there had been no return of the trouble.

(26) D. G., female, aged 24. September, 1908, to February, 1909: Looseness of upper front teeth commencing with ¹, which was extracted four years previously after mild attack of acute rheumatism; no improvement after six months' local treatment previously to September, 1908. June, 1909: General health improved; no sign of pus round teeth, though they were still slightly loose.

(27) W. C. B., male, aged 27, clerk. July, 1908, to April, 1909: Teeth became suddenly tender on June 28, 1908, and in three days were so loose and painful that he could not eat; he sought advice, but, getting no relief, came to Guy's on July 8. On that day all his teeth were loose and pus flowed freely;

42 Eyre & Payne: *The Bacteriology of Pyorrhœa Alveolaris*

gums thick, spongy and purplish, teeth coated with tartar; arthritic pains in both temporo-mandibular joints; skin a dusky pallor; temperature 100'4° F. Local treatment commenced at once, but extraction of fifteen teeth deferred for two weeks until the vaccine had raised patient's resistance to general infection. September 25, 1908: Better in every way, though suppuration continued in front of right mandible; several pieces of bone had been removed; denture inserted to replace extracted teeth. November 9, 1909: Patient feels perfectly well, gums and remaining teeth quite healthy, arthritic pains gone.

(30) W. M. B., male, aged 35, surgeon. October, 1908, to February, 1909: Acute pain, which started a few days previously in left upper wisdom and spread throughout upper and lower jaws; gums thick, turgid, and of a deep purple colour. November 9, 1909: Beads of pus can still be obtained by pressure around $\frac{11}{2,1}$; the rest of the mouth is quite clean and healthy.

The entire series of cases is shown in tabular form (*see* pp. 43-46).

Now a word or two as to the permanence of this cure effected by the administration of bacterial vaccines. All our cases that have been under treatment and have been discharged as cured have been requested to report themselves during the past fortnight, so that we might inquire into the present condition, but to our regret we have not been able personally to examine all of them. However, the result of our inquiries shows that "cure" has lasted in 7 from twelve to fifteen months, in 12 from nine to twelve months, and in 2 less than nine months; 4 were improved only.

TABLE I.—STAPHYLOCOCCUS AUREUS.

No.	Initials	Age	Sex	Teeth affected	DOSE OF VACCINE			Under observation	Result	Remarks
					No.	Size in millions	Interval			
1	A. R.	23	F.	All	5	50-100	14 days	67 days	Cured, 12 months	Rheumatism
2	E. M.	37	M.	All	9	50-250	7 "	60 "	" 10 "	Rheumatism

TABLE II.—PNEUMOCOCCUS.

No.	Initials	Age	Sex	Teeth affected	DOSE OF VACCINE			Under observation	Result	Remarks
					No.	Size in millions	Interval			
3	E. W.	39	F.	7,3 1,3,7 6,4,3,2,1 1,2,3,4,6	19	10-100	14 days	147 days	Cured, 12 months	Rheumatism
4	R. C.	52	M.	All	2	5-10	6 "	—	Died	Rheumatism; death due to malignant growth
5	B. C.	45	F.	All	6	10-100	9 "	50 days	Cured, 9 months	—
6	L. C.	19	M.	4,3,2,1 5,4,3,2,1 1	12	5-50	5 "	80 "	" 6 "	Rheumatism

TABLE III.—MICROCOCUS CATARRHALIS.

No.	Initials	Age	Sex	Teeth affected	DOSE OF VACCINE			Under observation	Result	Remarks
					No.	Size in millions	Interval			
7	R. M.	38	M.	$\frac{6,5,4}{4,3,2,1} \mid \frac{4,5}{1,5}$	6	250-500	14 days	62 days	Cured, 15 months	Rheumatism
8	J. C.	44	M.	$\frac{5,4}{1} \mid 7$	5	5-10	14 "	74 "	" 15 "	—
9	N. R.	30	F.	$\frac{4,3,2,1}{2,1} \mid \frac{1,2}{1,2}$	6	2-6-10	14 "	75 "	" 15 "	—
10	W. S.	43	M.	$\frac{4,3,2,1}{2,1} \mid \frac{1,2,3,4}{1,2}$	5	5-10	12 "	60 "	" 12 "	Rheumatism
11	H. H.	22	M.	All	—	—	—	—	—	Attended once only
12	R. K. B.	42	M.	$\frac{3,2,1}{5,4,3,2,1} \mid \frac{1,2,3,4,5,6}{1,2}$	4	5-25	17 days	2 months	Improved	Rheumatism
13	M. M.	54	F.	All	8	5-50	9 "	72 days	Improved	Rheumatism
14	J. S.	39	M.	$\frac{5,3,2}{5,4,3,2,1} \mid 1$	—	—	—	—	—	—
15	A. R. C.	36	F.	$\frac{3,2,1}{5,4,3,2,1} \mid \frac{1,2}{1,2,3,4}$	—	—	—	—	—	Rheumatism

1 New Cases.

TABLE IV.—STREPTOCOCCUS LONGUS.

No.	Initials	Age	Sex	Teeth affected	DOSE OF VACCINE			Under observation	Result	Remarks, constitutional symptoms, duration of cure
					No.	Size in millions	Interval			
16	R. L.	60	M.	5,4,3 1,2,3 7,5,4,3,2,1 1,2,3,6 1,2,3,4 4,3,2,1 1	9	50-250	14 days	123 days	Cured, 15 months	Rheumatism
17	F. H.	34	F.	4,3,2,1 1	5	5-25	21 "	88 "	" 15 "	Rheumatism
18	M. J.	53	F.	All	1	5	—	—	—	Only attended once; writes, November 9, 1909, "I have had all my teeth out and had new ones"
19	G. J.	35	F.	8,6,5,4,2,1 1,2,4,5,6,8 5 5	4	5	14 days	37 days	Cured, 10 months	Diagnosed tuberculous disease of jaw
20	F. C.	41	M.	All	9	5-50	9 "	84 "	" 12 "	Syphilis; lost 2 st. in weight; rheumatism
21	M. F.	37	F.	5,4,3,2,1 1 6,4,3,2,1 1	6	5-10	14 "	63 "	" 11 "	—
22	E. P.	18	M.	6,5,4,2,1 1,2 4,3,2,1 1	8	5-10	9 "	75 "	" 14 "	Rheumatism

TABLE V.—MICROCOCCUS CATARRHALIS AND STREPTOCOCCUS LONGUS.

No	Initials	Age	Sex	Teeth affected	DOSE OF VACCINE				Under observation	Result	Remarks	
					No.		Size in millions					Interval
					M.C.	Str.	Cat.	Str.				
23	W. H.	30	M.	All	3	2	5	5	14 days	30 days	Cured, 14 months	Rheumatism
24	M. M.	44	M.	— 1,2,3,4,8 7 3,4,5	2	1	5	10	11 "	33 "	" 12 "	Rheumatism
25	M. B.	24	F.	— 3,2,1 1,2,3	9	9	25	5-25	14 "	174 "	Improved, but not cured	—
26	D. G.	28	F.	4,3,2,1 2,3 — 3,2,1	18	5	5-10	10	7 "	158 "	Cured, 9 months	Rheumatism
27	W. B.	27	M.	All	23	6	50	50	14 "	272 "	" 7 "	7,6 4,5 Extracted 7,5,4,3,2,1 1,2,3,4,7
28	F. M.	43	M.	5,4,3,2,1 1,2,3,4,5,8 — 3,2,1 1,2,5	6	2	5-10	10	7 "	60 "	" 11 "	Rheumatism
29	B. O.	25	F.	5,2,1 1,2,3 — 5,4,3,2,1 1,2,3,4	4	6	5-10	5-10	15 "	150 "	Improved	Two central lower incisors must come out; others all right
30	W. M. B.	35	M.	All	9	6	25-50	5-10	10 "	—	Improved	—
31 ¹	C. H.	36	M.	4,3,2,1 1,2,3 — 4,3,2,1 1,2,3	—	—	—	—	—	—	—	—
32 ¹	M. G.	17	F.	— 4,3,2,1 1,2,3,4	—	—	—	—	—	—	—	—
33 ¹	H. H.	33	M.	All	—	—	—	—	—	—	—	—

¹ New Cases.

A Case of Unilateral Overgrowth of the Lower Jaw.

By STANLEY BOYD, F.R.C.S.

Miss D., aged 21, was brought to me by Mr. Douglas Gabell, complaining of facial deformity. This is made up of the following elements: The chin is prominent and directed to the right. The left half of the face from the malar to the lower edge of the mandible is much longer than the right, and the lower border of the left mandible makes a heavy curve downwards between the symphysis and the angle. The lower edge of the right mandible is convex outwards, and gives too great a prominence to this region. The mouth is asymmetrical, especially in action, and this is obviously due to the greatly increased depth of the left mandible below the angle of the mouth, which renders the lower lip too short easily to meet the upper. Thus the patient can often be seen sitting with the left halves of the lips considerably separated. If the tissues of the chin and cheek are gently pressed upwards, so that the lip is freed from the downward traction of the jaw, its movements at once become normal. The mouth opens freely and smoothly; there is no obvious swelling over the temporo-maxillary joints. The front teeth are of poor quality; all the upper incisors and left canine are loose and tender; the gums are retracting. The bite, as regards mere alignment, has been wonderfully preserved, the teeth in the right mandible having all acquired a slope towards the mid-line, but the lower molars touch the uppers only by their outer edges. The gap between the lower centrals is directed upwards, and to the left of that between the upper centrals. All four first molars have been removed, and this on the right side has facilitated the displacement of the second and third lower molars noted above. Measurement of the mandible gives the following results: From condyle to angle, left 3 in., right 2 in.; from angle to symphysis, left 4 in., right $4\frac{1}{4}$ in., measured along the curve in each case. The fingers show that the left condyle is enlarged and that it goes back far in the glenoid fossa; the enlargement does not seem to be great. The left angle is open and rounded. In front of the angle the lower border becomes very thick and down-curved, whilst the right lower edge is also thick and convex outwards. The left mental tubercle forms the "point" of the chin. The right angle, ramus, and condyle appear to be normal. The depth of the tooth-bearing portion of the left mandible is greatly increased.

The history is that the deformity began at about 14 years of age with

a "dropping" of the left mandible—i.e., projection downwards of its lower edge. Then the chin deviated to the right, and slow progress has since been maintained in both directions. No cause is assigned. The patient was very rickety, and did not walk till she was aged 3. Her first teeth were mere shells. At the age of 3 she knocked out both upper centrals in a fall from a carriage. When aged 8 she struck the left side of her face against a fence in a fall from a swing; but no severe pain and no bruise resulted. As long as she can remember she has suffered much from toothache. At the age of 13 she had three abscesses apparently connected with some left lower tooth; one was opened through the skin $\frac{1}{2}$ in. below the left angle of the mouth, the others burst into the mouth. Except measles she has had no illness requiring treatment. Her joints and bones in general appear to be normal.

Everything in this case seems to place it in that small group (about nine recorded) characterized by enlargement in the neighbourhood of one or other condyle of the mandible; almost all the cases have been treated by excision of this condyle, and the excised portions have all shown more or less enlargement by the addition of true cancellous bone covered by the usual dense surface lamina, often with marked irregularity of the surface of the bone, and also of the articular surface. The largest condyle was found in Heath's case— $1\frac{1}{4}$ in. by 1 in. The newly developed bone is absolutely continuous with that of the ramus. Some have regarded the overgrowth as a sort of diffuse osteoma; others as inflammatory; others, going further, have attributed the changes to rheumatoid arthritis. In only one case was rheumatoid arthritis of other joints present. I do not know of lengthening of other bones produced by rheumatoid arthritis, nor have I any clear conception of how lengthening of the neck of the adult jaw is produced. I presume that it must be by direct ossification of the abnormal tissue capping the condyle in these cases. But given the increased length of one ramus, the changes I have described in the arch of the mandible, affecting primarily, as they do, its strain-bearing portion or base, are probably not due to disease but to muscular effort trying to preserve the bite, and this effort has been more successful than usual, because it was made upon a still growing mandible.

With regard to treatment, it is clear that removal of the left condyle and neck will not efface this deformity, and particularly not the deformity of the lip. The bite will, I should imagine, be altogether thrown out. I should greatly appreciate the advice of this Section as to the best way to deal with the teeth. Should those in the left mandible be

removed to facilitate return of the jaw to the normal position and not be replaced by a denture, so that the left jaw may atrophy and yield more readily to modelling force? How long are these likely to take to produce a reasonably good cosmetic result? Should the lower edge of the left mandible be excised? These are questions I would gladly hear answered.

DISCUSSION.

Mr. J. F. COLYER said he had had an opportunity of seeing the case two days ago, and he did not see how a good result was to be obtained unless the teeth were removed. As he had pointed out at the time, a good many teeth already showed signs of periodontal disease. When he saw the case it seemed to him a little doubtful whether simply removing the condyle would help the mandible to move up sufficiently, and he suggested to Mr. Boyd that he should remove sufficient of the ascending ramus to allow the superior border of the mandible on that side to come up to a higher level than that on the opposite side. By this means a better result from an aesthetic point of view could be obtained.

Mr. STANLEY MUMMERY thought the removal of the teeth would not quite correct the deformity, because the muscles were elongated on that side, and until they had contracted there would be no tendency for the cut end of the jaw to be drawn up into the socket. It seemed to him that some form of skull-and-chin cap would be necessary to hold the mandible in position until the muscles had contracted sufficiently to hold it by themselves.

Mr. STURRIDGE said he had examined the mouth and noticed that the teeth in the superior maxilla were very much lower on one side than on the opposite side, probably the depth of half the crown a tooth lower on the affected side than on the normal side. It would be impossible to do anything without removing those lower teeth and also the alveolus, and, to bring it nearly to a level, a very large operation on the condyle would be necessary as well.

Mr. MOUNTFORD said it seemed to him, if any teeth were extracted at all, it would be much better to remove upper teeth, because dentists could far more satisfactorily replace lost upper teeth than lower. He was a little surprised to note that Mr. Boyd considered the condition was due to overgrowth of the condyle. He would have expected to see the articulation of the teeth very much more disarranged than it was; in fact the teeth on the affected side articulated better than on the other. It certainly seemed a very curious case. There appeared to be some loss of tissue on the right side, and the whole jaw seemed as if it might have been slewed round by the muscles being stronger on one side than on the other. It seemed to him, as the last speaker had pointed out, that, the upper teeth being on a lower level on one side, it would be far better to remove the upper teeth than the lower. It was a question of re-arrangement of the articulation of the teeth, trusting to get a good result.

Mr. H. LLOYD WILLIAMS said the case was an extremely interesting one. It was not the alveolar bone so much as the base of the mandible that seemed to be hypertrophied. The skiagram showed that the lower portion of the jaw seemed to be very much overgrown. If that was the case, and taking it also that the condyle was lengthened, he thought any extraction of teeth and removal of the condyle without dealing with the base of the lower jaw itself would certainly not cure the deformity. The bite now was quite good. Whether, as had been suggested, the original irritation which caused the growth was due to chronic septic periodontitis was also open to doubt, because the new growth included the ascending as well as the horizontal ramus. He quite agreed with Mr. Sturridge that the upper teeth were much lower on the left than on the right side, but that might be explained by the fact that the condyle had increased in length, and that there was a compensatory effect in the maxilla to preserve the articulation of the teeth.

Mr. F. J. BENNETT advised Mr. Boyd to follow the plan that was often adopted in cases of fractured jaw—namely, to take a model of the upper and lower jaw and all the portions concerned, saw the model up in various ways and put it together again in as nearly an ideal condition as possible. It was wonderful what a number of hints one obtained from that procedure.

Mr. STANLEY BOYD, in reply, thanked the members for the kind way they had considered the paper and given him the benefit of their experience. He had been very interested in Mr. Sturridge's remarks, as he had not noticed the downward curve of the left upper jaw. It was a very interesting thing, and emphasized the point which he laid stress upon in the paper—namely, the extraordinary way in which the bite had been preserved. Clearly, if, as he believed to be the case, the left ramus was elongated, the lower teeth would be removed from the upper, and the upper would therefore be unopposed, and a downward growth would take place. That seemed to him to be what had happened. The lower jaw did what it could to meet the defect, and the upper helped out. With regard to the removal of the teeth, he gathered that the majority of those who had spoken were in favour of removal of the teeth in the left mandible; some apparently would trust to that alone, while others would add the removal of the alveolus. But he did not think even this would be sufficient; for measurement showed the left ramus to be about 1 in. longer than the right. The primary trouble was undoubtedly below the left condyle; so this would have to be dealt with. Mr. Mummery suggested that if the condyle and teeth were removed, the muscles, being elongated on that side, would fail to bring the jaw back into position. He did not think that would be the case. The muscles, being strong and active, would very soon draw the jaw up and back. Upon the question of removing teeth, Mr. Mountford suggested that the upper teeth should go because they were the ones most easily replaced. Mr. Boyd thought there would be but little room, when (if ever) the change had taken place, for teeth of any size upon the left side; in all probability there would be room only for a chewing block. As the removal of the teeth in the lower jaw would allow the jaw to move freely on the upper,

would tend to produce atrophy of the lower jaw, and would thus favour its re-modelling, he thought he would be in favour of removing the lower teeth, as Mr. Colyer first of all suggested. Mr. Bennett's advice—to make a model of the jaw, cut it up, and see what could best be done with it—would often be very useful; but here disease in the neighbourhood of the condyle fixed the treatment. He thought the best thing would be to suggest to the patient removal of the condyle and the teeth, and then wait and see what the result was. If she still retained the arching down of the left mandible, that could be removed later on. He had also been very much interested in Mr. Lloyd Williams's remarks, and was in general agreement with them.

A Case of Fracture of the Mandible set with a Silver Splint made by the Casting Process.

By C. SCHELLING, L.D.S.

ALTHOUGH I am aware that there are practitioners who can, as the result of large hospital experience, satisfactorily put up a fractured mandible with a few pieces of iron wire in a very short time, yet I trust that the case I am about to describe may not be without interest to those persons who are liable at any time to be called upon in the expectation that they will be able to render material aid to the medical practitioner in charge of such a case.

On the evening of April 28, 1909, I was sent for by Dr. G. C. Ouseley, of Blackheath, to see a patient of his, a gentleman aged 41, who, when slowly cycling along behind a tramcar, was run into by another cyclist, of the scorching type, who suddenly came round the car in the opposite direction, and appears to have struck the patient on the right cheek with his head and disappeared. The patient did not lose consciousness, but directed a cabman to drive him home. On the way he obtained crepitus, and also diagnosed a dislocation on the left side, and from the great displacement of the parts considered himself to have lost a tooth, and regretted having left it behind. The fracture was compound into the mouth and considerable hæmorrhage took place. On his reaching home Dr. Ouseley was sent for and promptly applied a bandage, and attempted to find me by telephone, but it was not until two hours later that I arrived.

The fracture was vertical through the symphysis, something like crepitus was also found somewhere near the left angle, and if any dislocation had taken place it had been reduced. The parts of the

jaw were considerably separated, but no tooth was missing. After weighing the comparative advantages of immediately wiring the jaw-bone or of fitting a dental splint, the latter was decided upon, and impressions of the upper and lower jaws taken in composition. Cheek pieces of composition were also squeezed in with the jaws held together with the fingers, and were found of great use later. The models were cast, and the lower one divided at the seat of the fracture and fitted to the cheek pieces and upper model, and reunited with more plaster.

Next day I was designing a splint to be made of wire in the workroom when our chief assistant suggested that the new casting arrangement might be of use, so the splint which I show was cast in silver in one piece, the lugs being so placed as not to interfere with the patient's bite. That evening I, after a good deal of difficulty, got the splint into place, and once there it gave a sense of comfort to the patient, who was, however, considerably fatigued by the next part, the wiring of each tooth to the inner bar of the splint, as the slightest pressure directed towards the left condyle gave great pain. All the ligatures were lightly twisted at first, and, finally, all tightened up, the ends cut off pretty short and then tucked away under the outer bar. The teeth were so crowded that only the finest binding wire could be pushed between them. When the wiring was concluded the other crepitus could no longer be felt, and was attributed to some damage in the articulation; but as the mouth could easily be opened widely the idea of sending the patient to town for a skiagram was not carried out. On the third day the right-hand end of the splint could be lifted by the tongue, and needed re-wiring.

Dr. Ouseley saw the patient twice daily for a fortnight, and a nurse was installed, with instructions to see the mouth well cleansed with sanitas and carbolic-acid lotion every three hours, night and day, and to this, and to the patient's great previous attention to the hygiene of his mouth, we attribute the fact that at no time was any pus or fœtor apparent.

In six weeks the splint became loose, and mastication, after one cusp had been slightly reduced, was perfect. Three visits were all that I found it necessary to pay, and the patient, who is himself a licentiate in dental surgery, is present, and has kindly expressed his willingness to answer any questions, and to allow anyone who wishes to do so to examine his mouth. The absence of any callus is, I believe, an indication of the close apposition in which this simple arrangement held the parts until union was established.

(The patient was present and was examined by the members.)

Odontological Section.

January 24, 1910.

Mr. WILLIAM HERN, President of the Section, in the Chair.

The PRESIDENT announced that before Dr. Eyre and Mr. Payne's paper (read at the last meeting) was discussed, Mr. Payne would add some supplemental remarks and Mr. Kenneth Goadby would read a paper on "The Vaccine Treatment of Early Cases of Pyorrhœa Alveolaris"; the discussion would then be taken on the three communications.

Mr. J. LEWIN PAYNE: In order to facilitate discussion, perhaps I may be allowed to briefly summarize the paper which was read at our last meeting, and then to add one or two points concerning the clinical aspect of the cases referred to. At first sight it might seem that the examination of pus in the disease commonly called pyorrhœa alveolaris would at once afford valuable information concerning its ætiology, but the pus obtained from the sockets of affected teeth is teeming with representatives of an almost incredible number of different species of bacteria, and it is impossible to propagate even one-tenth of these upon nutrient media under the artificial conditions of the laboratory. None of the organisms hitherto detected in the pus can be regarded as specific, but Sir Almroth Wright's work on immunity has provided a means for determining whether or no any given organism plays an active part in the pathological condition with which it is associated. Wright has demonstrated the presence in the blood-serum of bacterial antibodies which he has named opsonins. Opsonic indices, as they are called, are expressed in percentages of unity, and in the normal individual variations occur between 0.8 and 1.2, an index below or above these extremes being held to indicate infection with the organism in question. The results of treatment with vaccine also serve as a guide. Having determined that the patient is affected with a particular organism, an attempt is made to immunize the individual by injecting killed cultures of that organism into the patient in such a manner as to increase the

amount of the available antibody. If the organism employed in the injection is actually the one responsible for the pathological condition, an excess of opsonin will be provided and will lead to an improvement in the clinical condition of the patient. Mr. Goadby pointed out that micrococci of the various groups were the responsible organisms for these cases of so-called pyorrhœa alveolaris, and by the administration of vaccines, prepared sometimes from one organism and sometimes from a mixture of several bacteria, he has shown that it is possible to eliminate this form of suppuration. Attention has been drawn in the paper to the fact that in the specimens of pus derived from different patients the organisms constantly present were cocci, spirillæ, and fusiform bacilli, and of these only the cocci appeared in the cultivations obtained from the pus. Of those cocci which could be isolated six species were readily recognized—the *Streptococcus brevis*, the *Streptococcus pyogenes longus*, the *Streptococcus lanceolatus*, the *Micrococcus catarrhalis*, the *Staphylococcus aureus*, and the *Staphylococcus albus*. Washbourn and Goadby have proved that the *Streptococcus brevis* is only a harmless saprophyte, but the five remaining cocci are well-known pathogenic micro-organisms, and it seems probable that one or more of these may be responsible for the form of suppuration we are discussing. We do not consider pyorrhœa alveolaris to be a specific disease in the sense that it is due to an infection by one particular micro-organism, but we do regard it as a suppurative process due to one, or more than one, of the ordinary pathogenic bacteria, a disease which owes its character and course to the accident of its anatomical position. The vaccine treatment has, we believe, been attended with a larger measure of success than can be accounted for by mere coincidence. I need not again refer to the methods employed beyond saying that in identifying the responsible organism the patient's blood was tested against each of the micrococci in turn. The organism or organisms to which the opsonic index has been particularly low, say 0·5 or lower, or particularly high, say 1·3 or higher, were regarded as responsible for the infection. Out of 33 cases the following organisms were isolated: *Micrococcus catarrhalis* in conjunction with the *Streptococcus pyogenes longus* in 11, *Micrococcus catarrhalis* in 9, *Streptococcus pyogenes longus* in 7, *Streptococcus lanceolatus pneumoniæ* in 4, and *Micrococcus pyogenes aureus* in 2.

In regard to the clinical aspect of these cases I should like to emphasize one or two points. In the first place, they were not ordinary cases of pyorrhœa alveolaris; they were specially selected, either on account of their severity or because the ordinary local treatment, after a fair trial, had failed. It may be possible to cure the simple cases of pyorrhœa alveolaris by means of local treatment alone, but in nearly all of those contained in the tables shown the treatment had been undertaken as a last hope, when it appeared that most of the teeth would have to be sacrificed.

Another point of considerable interest is that in a large number of these

cases muscular or joint troubles were complained of, and described by the patient as "rheumatic" pains. In 24 out of 38 patients these so-called rheumatic pains were present; 7 out of the 24 had definite signs of osteoarthritis, and, what is perhaps of greater interest, these so-called rheumatic pains disappeared in nearly every instance after the vaccine treatment was commenced.

Although the subject which we are specially considering in this paper has to do with the use of vaccines, and whilst the treatment of the cases in our series has been primarily carried out on these lines, we have no intention of suggesting that the local surgical treatment should be dispensed with. On the contrary, we deem it important that the teeth should be cleansed, scaled, treated with antiseptics and the application of splints, &c., in addition to the treatment of the gums and the alveolar margins, which should be scarified and cauterized when necessary. We do not, at present, suggest that the vaccine treatment should be adopted for every patient who is suffering from pyorrhœa alveolaris, nor is it maintained that the condition can always be cured; but in all cases of extreme severity and in those showing constitutional manifestations of the disease, or in those where the local treatment has failed to effect a cure, we believe that the dental surgeon will obtain the best results if the vaccine treatment is employed.

The Vaccine Treatment of Early Cases of Pyorrhœa Alveolaris.

By KENNETH W. GOADBY, M.R.C.S., L.D.S.

IN the early part of last year our Secretary suggested I should give some review of the treatment of alveolar pyorrhœa, or alveolar osteitis, which would be of interest from the points of early diagnosis and treatment. I have therefore chosen the question of early pyorrhœa rather than the later stages, when the disease is so far established that, although the diagnosis is a simple matter, the treatment is by no means so satisfactory.

As you are aware, alveolar osteitis has engaged my attention for a considerable time. As early as 1906 I had the extreme good fortune to be associated with the late Dr. J. W. Washbourn in some preliminary work with regard to the streptococci of the mouth, and from that date I have continued my researches uninterruptedly. In my earlier investigations, given in the *Transactions of the Odontological Society*, various

mouth bacteria were described—having general significance rather than special relation to any one disease, but in 1904 I published an account of some experiments which showed that by inoculating the material from alveolar diseases into animals I was able to produce some of the general symptoms observable in man, but that attempts to reproduce the disease in the gum margins were not successful. I suggested then that “the potentiality for disease of oral sepsis received considerable support from even a preliminary survey of its bacteriology, perhaps pointing to its operation in an even wider pole than is at present credited or admitted.” A little later, in a paper read in 1905, I called attention to the general symptoms associated with disease of the gums, and gave in addition the opsonic index of certain of the organisms which I had isolated from the cases. I pointed out then the value of the opsonic index method in determining which of the bacteria obtained in cultivation were the cause of the disease, and I gave a table of results of eleven cases of pyorrhœa alveolaris which had been treated by means of vaccines, and which had improved not only in the general, but in the local symptoms as well. In the conclusion of that paper I pointed out the fact that the disease is not a mere local one; that the injection of vaccines produced amelioration of the general symptoms as well as the local disease, and that the vaccine method, if applied properly, opened up a new field of treatment. In 1906 I gave another series of eighteen cases of staphylococcal pyorrhœa which had been treated by means of staphylococcic vaccines. I also published then the photographs of two of the organisms other than staphylococci which are often present in the deeper layers of the infected bone. In the Erasmus Wilson lecture in 1907 I gave a tabulation of a large number of cases, both of their bacteriological findings and their method of treatment, and the results therefrom.

I find, in looking over my records, that I have had the opportunity of making bacteriological examination and blood tests in 324 cases of pyorrhœa alveolaris of varying intensity, some in the very early stages, others in the final stage, and a few during what proved to be a terminal fatal illness. I find that in these 324 cases, eighteen had serious complications directly referable to the mouth disease, ten of them proved fatal—three from epithelioma commencing in the gums, and seven from septicæmia or meningitis; four other cases also suffered from lymphatic leukæmia.

For clinical purposes pyorrhœa alveolaris may be divided into three main groups:—

(A) EARLY CASES.

(1) Where the gums and teeth appear perfectly normal, but here and there slight depressions may be noticed between individual teeth, and careful examination in these regions reveals, sometimes only microscopically, the presence of pus with the cells old and degenerated, indicating that the process is slow; a large number of different bacteria are present. An X-ray photograph, in such an instance, shows that the interdental bone has already become infected.

(2) Easily bleeding gums slightly detached from the teeth, increase of the gum tissue of the interstitial spaces, and a small amount of pus, often only recognized microscopically.

(B) CHRONIC FORM.

Hypertrophied gum, or later atrophic. Well-marked discharge from the sockets of one or more teeth. Microscopically the pus full of well-formed pus cells, many of which are loaded with bacteria; the presence of large numbers of bacteria in the liquor puris, loss of the alveolus, not only between the teeth, but in the anterior and posterior alveolar plates, and patches of rarefying osteitis here and there about the alveolus, which are well seen in an X-ray photograph. Infected mucous glands, which I have described elsewhere, are to be seen near the infected areas.

(C) LATE.

All the teeth are loose in their sockets; alveolar process largely absorbed; gums boggy, and sinuses here and there to be found leading down to necrosed bone or bare roots. Small nodules, marking the positions of cicatrices of old sinuses, may also be seen here and there about the gum.

In *A* and *B* the teeth may be saved, in *C* but rarely; whilst the removal of all the teeth in the type *C* by no means always removes the general symptoms. After extraction the general symptoms become greatly improved in most instances, but the local disease of the bone frequently remains. As is well known amongst dental surgeons, difficulty exists of accommodating artificial dentures in persons who have had a large number of teeth extracted for alveolar pyorrhoea,

the dentures "sinking" and requiring constant altering and adjustment, owing to the progressive rarefaction of the bone.

This brief outline of clinical facts is necessary to define the cases I am describing. For the present purpose I considered it was better to confine my attention to those cases of type *A*, in which the local symptoms were ill-defined and often unrecognizable, except by bacteriological and microscopical examination. I have therefore gone over my records, and tabulated all the cases of early pyorrhœa in which I had been able to make a complete bacteriological examination up to July, 1909. There are in all seventy cases, eighteen of which only have been previously reported upon. In all instances practically all the bacteria present were obtained in pure cultivation, the exceptions being the spirochætes commonly present in the mouth, and some of the thread forms. Most of the other organisms, however, I have now been able to grow, but only those organisms are scheduled which showed an abnormal opsonic index. All the organisms isolated were so tested, and those giving an abnormal opsonic index were selected as the ones directly concerned with the disease. This method, as I pointed out in 1905, I have found gives reliable and satisfactory results, and is, I find, the best method at present possible to determine the infecting organisms.

Of the seventy cases I am discussing, twenty-one had no general symptoms of any description, although two of them suffered from recurring attacks of ulceration of the mucous membrane of the mouth. Of the remaining forty-nine, twenty had some slight general symptoms, such as general malaise, slight gastro-intestinal disturbances, and some slight signs of toxæmia. In the remaining twenty-nine cases definite signs of general infection were found, including rheumatic symptoms, colitis, loss of weight, and malaise, particularly on exertion.

TABLE A.—BACTERIA FOUND INFECTING 70 CASES OF EARLY PYORRHOEA ALVEOLARIS.

	(A) No general symptoms	(B) Slight symptoms	(C) Definite symptoms	(D) Total
<i>Streptococcus longus</i> ...	3	4	12	19
<i>Micrococcus catarrhalis</i> ...	5	9	9	23
<i>Pneumococcus</i> ...	—	1	4	5
<i>Staphylococcus aureus</i> ...	2	2	8	12
<i>Micrococcus citreus granulatus</i> ...	—	6	—	6
<i>Bacillus necrodentalis</i> ...	11	3	—	14
<i>Bacillus septus</i> ...	5	4	6	15
<i>Saccharomyces neoformans</i> ...	—	2	3	5

The bacteriology of the cases is interesting. Reference to the tables shows: (1) That the pathogenic bacteria of the coccal group are more frequently present when general symptoms are found; (2) The *Bacillus necrodentalis* is more common in the quite early cases with no general symptoms, and that an organism (*Bacillus septus*) belonging to the so-called Diphtheroid group is often associated with the disease. The *Saccharomyces neoformans* did not occur in the first series of cases.

TABLE B.—RELATION OF GENERAL SYMPTOMS TO BACTERIA FOUND IN ALVEOLUS.

	Rheumatic	Anæmia	Gastro-intestinal	Other	Total
<i>Streptococcus longus</i>	... 8	... 1	... 2	... 5	... 16
<i>Pneumococcus</i> 1	... 2	... 1	... 1	... 5
<i>Staphylococcus aureus</i>	... 1	... 4	... 5	—	... 10

Turning now to the symptoms related to the bacteria found, and taking the whole of the cases together which showed general symptoms, I find that rheumatic symptoms occurred in ten cases—eight times associated with the streptococcus, once with the pneumococcus, and once with the staphylococcus. Gastro-intestinal symptoms occurred twice with the streptococcus and four times with the staphylococcus. Loss of weight occurred ten times amongst the staphylococcal cases, but only twice amongst the streptococcal cases and once with the pneumococcal. Recurrent quinsy was also associated with staphylococcal infection, and a case of recurrent vomiting was associated with the pneumococcus.

What I wish particularly to emphasize as the result of these observations is that early infection of the alveolus with certain of the pathogenic organisms, particularly the streptococcus, pneumococcus and staphylococcus, takes place in an insidious manner, and may give rise to symptoms out of all proportion to the amount of local infection.

From the association of the *Bacillus necrodentalis* and the *Bacillus septus* and the *Micrococcus catarrhalis* with the early cases, one or other of these organisms occurring in every one of the early cases, points to one or other of these organisms being concerned in the early stage of infection, particularly as common coryza is often associated with sore gums. It is possible that in a number of instances spontaneous cure of an early pyorrhœa takes place, particularly when the infection is of the nature of a general gingivitis, and it is this class of case of general gingivitis which is particularly amenable to local treatment. A further suggestive point is that the opsonic index in such cases is frequently at a

60 Goadby: *Vaccine Treatment of Pyorrhæa Alveolaris*

very high level, showing that considerable reaction on the part of the body towards the elimination of the disease is taking place. Instead of a spontaneous cure taking place, or of a cure due to local treatment, interstitial infection may continue so that the disease progresses quietly but insidiously. Simple gingivitis is more common in young adults and children than in adults. A considerable percentage of individuals showing the latter stages of alveolar absorption and loosening of the teeth give a history of chronic gingivitis, from which they suffered whilst in their teens.

TABLE C.—RESULT OF TREATMENT OF 70 CASES OF EARLY PYORRHÆA.

	Cured	...	Relieved	...	Disappeared	...	Died	...	Total
(A) No general symptoms ...	15	...	2	...	4	...	—	...	21
(B) With general symptoms	30	...	11	...	7	...	1	...	49

TREATMENT.

The methods used in the treatment of my cases are threefold:—

(1) The inoculation by means of bacterial vaccines made from the organisms isolated from the individual case and injected subcutaneously in doses of 50 to 250 millions at a time, the number of inoculations required ranging from five to fifteen, at intervals of ten to twenty days, and controlled by opsonic estimations.

(2) The use of local treatment, including lavage and syringing, surgical interference where indicated, and the extirpation of any pockets by means of the actual cautery. This is best done after preliminary immunization.

(3) Medicinal treatment where indicated, arsenic in some form being particularly useful; in cases with anæmia, iron and arsenic.

For the treatment of the cautery wounds, syringing with 1 per cent. sodium citrate in normal saline gives excellent results, if immunization has been carried out.

The foregoing methods have been used in these seventy cases. Forty-five have been cured; the discharge has ceased, the alveolus has become hard again, and the general symptoms have disappeared. In thirteen relief has been obtained—four of the cases went away before the treatment was completed, and the other nine have remained permanently relieved, but not entirely cured. Eleven other cases disappeared during the course of treatment, but these were mainly hospital cases. One case died.

The diagnosis of early alveolar pyorrhœa or alveolar osteitis should be bacteriological and microscopical, and when any deficiency is found in the interdental bone, a skiagram should be taken, the photograph often revealing early bone infection, which mere inspection does not show. Should any infection be found to exist and bacteriological examination show that organisms are present to which the patient's serum gives a low resistance, immunization by means of appropriate vaccines should be undertaken. The prognosis in early cases is over 60 per cent. of cures; whilst without bacterial treatment the majority will progress not only to loss of their teeth, but to disease, of diverse natures, which have been shown to be associated with pyorrhœa alveolaris.

The Bacteriology of Pyorrhœa Alveolaris and the Results of Treatment by Bacterial Vaccines.

DISCUSSION.¹

Mr. KENNETH GOADBY, in opening the discussion, said he had given a good deal of attention to the subject of the bacterial treatment of pyorrhœa alveolaris and had spent a considerable amount of time in research work on the subject. He felt particularly grateful to find that a subject that up to the present had been confined almost entirely to people working like himself should have at last come within the range of one of the acknowledged lights in the bacteriological world in London, his old friend Dr. Eyre; and he was much gratified that his own work had received such ample confirmation. He did not think, on the whole, that the bacteria of the mouth had received the attention they should have received, and the work of the pioneers had been to some extent overlooked, although such work always formed the starting point for other investigators. There were three main points on which he was by no means in entire agreement with Dr. Eyre and Mr. Payne. He did not at all agree that the best method of attempting to find out the particular specific bacteria related to any given disease was to make a special selection of the micrococci, confining one's attention to the estimation of the opsonic index only with regard to the micrococci present. He had found in his earlier work, in which the opsonic index was estimated for a large number of organisms present, that if the estimation had been confined to any one division, say micrococci, a number of them might be found to be related to the disease itself, but that some of the bacilli

¹ Adjourned from November 22, 1909.

62 Discussion on the Bacteriology of *Pyorrhœa Alveolaris*

as well, when tested, also gave a low index. If the opsonic index had any value at all, if it showed a diminution or a large increase of any one organism, well and good; but if a large number of organisms were related thereto, the chances were either that the opsonic index did not give all the knowledge required, or the other organisms were also concerned in the infective process. He had shown that evening that some of the bacilli which had been rather looked askance at by Dr. Eyre were concerned, at any rate, in the early stages of the disease. Then with regard to the Gram-negative cocci in the mouth, his own experience did not correspond with that of Dr. Eyre. He certainly found there were a number of Gram-negative cocci in the mouth, which might be found present with other bacteria, and giving a low index towards the patient's own serum, but that a number of these micrococci did not by any means conform to the commonly accepted type of the *Micrococcus catarrhalis*; but for convenience of general work, it was well to class them as such. He would like to know whether all the *Micrococci catarrhales* which Dr. Eyre described gave absolutely the tests of absence of fermentation on carbohydrates besides being Gram-negative, and so forth. Thirdly, with regard to the question of general and constitutional symptoms and infection, for some years past he had examined cases of pyorrhœa, and the pathological conditions related thereto, and he did not feel that the general symptoms produced could be directly attributed to the mere swallowing of fetid pus. He thought the general symptoms, such as toxæmia, septicæmia, acute intoxication, and so on, could scarcely be attributed to the swallowing of minute doses of bacterial products, but were due to invasion by bacteria and bacterial products (toxins) into the blood-stream direct. The amount of fetid material that could be obtained from the mouth and that which might exist in any healthy colon would not differ materially, although, of course, the absorption in the lower parts of the intestine was smaller than higher up. He had found in a number of cases bacteria present in the blood, and he certainly could not agree with Dr. Eyre that the chief cause of the symptoms produced in cases of pyorrhœa was merely the swallowing of small quantities of fetid pus.

Mr. STURRIDGE, while thanking the three authors for their work, wished he could hold the opinion that at last a permanent cure had been found for pyorrhœa. He could not help thinking that Dr. Eyre must have had a very limited experience of the disease to allow himself, after a brief term of a year or eighteen months, to say that the cases of pyorrhœa he had treated were cured. Dentists who had had twenty years' or more experience in the treatment of the disease would appreciate his objection to calling those cases cured. Many cases of pyorrhœa treated by the extraction of the loose teeth, scaling of the remainder, the use of a good mouth wash, and thorough cleanliness, would improve to the extent Dr. Eyre had mentioned, and would remain improved for eighteen months, especially where patients had never been treated before; but the cases

were not cured. He should like to hear from Dr. Eyre what his conception of the ætiology of the disease was. Did he consider the presence of micro-organisms to be the cause or the effect of pyorrhœa? Did he in his treatment ignore all such causes as malocclusion, inter-spacing, deep-seated salivary calculi, and shifting of the teeth in their normal positions? If, for instance, teeth were elongated and were being forced out of position at every closure of the jaw, did he expect vaccine treatment to cure such teeth? He felt sure that vaccine treatment had a useful place in connexion with pyorrhœa, but he was equally sure that no permanent good results could be expected if energies were relaxed in the direction of recognizing and removing all direct local causes. Local treatment must be kept up during the vaccine treatment, and pursued for a very long time after. For a long time, by a method of ionic medication of the periodontal membrane by electric current, he believed he had been producing very similar results in the system to those claimed by the advocates of vaccine treatment. Ions of antiseptic salts were driven into the very protoplasm of the affected tissues and killed the organisms in the tissues, and he took it that the dead organisms were absorbed into the blood-stream and had an effect upon the opsonins similar to that of vaccines prepared from cultures. The method was simple and surer than vaccines, in that there could be no failure in procuring the correct organisms. The effect of checking the pus was far quicker than that reported by the vaccine treatment. Any dental practitioner could undertake the work, and he had had patients who had no recurrence or loss of any of the teeth ten years afterwards. Nevertheless, experience had taught him not to say that pyorrhœa cases were cured. With respect to Mr. Goadby's paper, it only went further to confirm the opinion that local causes of the disease were being overlooked, while the bacterial effects of the disease were being pursued to an extent which was likely to lead men astray. The cases he had examined that evening went further to confirm that opinion, because they showed extensive extraction of loosened teeth, and the teeth left were not thoroughly scaled. He thought the extraction of teeth, and the thorough cleaning of the remaining teeth would have brought about the same improvement without any vaccine treatment.

Mr. J. G. TURNER said that, as far as the first paper was concerned, he was in agreement with Mr. Goadby, and could only echo what he had said. With regard to infection via the gum, the temperature was sufficient to show it, as the temperature in gastric cases took much longer to rise than that due to local absorption. He joined with Mr. Goadby in thanking Dr. Eyre for taking up the subject, as he found the general bacteriologist was generally afraid of the mouth. While talking of Dr. Eyre's paper he might answer one point in Mr. Sturridge's remarks by saying that he had never yet seen a case of pyorrhœa, local or general, that he could ascribe to malocclusion. He had seen one that made him think it might be so, but on examination he found

64 *Discussion on the Bacteriology of Pyorrhœa Alveolaris*

there was nothing of the kind. Pyorrhœa was wholly due to local infection, and could be discovered at a much earlier stage than that Mr. Goadby had mentioned. What he himself called an early stage was the first trace of gingivitis, which, if left alone, would inevitably end in pyorrhœa. Such cases examined bacteriologically showed as many forms of micro-organisms present as would be found ten years later when the person was suffering from poisonous pyorrhœa. Around the first erupted permanent teeth of children aged 7 and 8 could be seen the beginnings of pyorrhœa, and unless those children were treated by the age of 15 or 20 they would have fully developed pyorrhœa. The effect of local treatment in early pyorrhœa was to prevent the development of the disease; and absolutely nothing more was needed than local cleanliness, the cleanliness extending, if need be, to the extraction of the teeth. Clean the teeth thoroughly, clear out the pockets, using none of the pretty-looking mouth-washes, but cleaning by actual rubbing and swabbing; if that treatment were thoroughly carried out, within a fortnight of seeing a patient who was pouring with pus the gums would look almost pink. But, although no pus might be passed, the man was not cured, and the disease was going to recur unless local treatment were very carefully continued. With regard to vaccine treatment, he had had the opportunity of seeing three specially selected cases of Dr. Eyre's, and he would not call them anything very special, and he would not call them cured. All of them presented inflammatory reaction round the edges of the gum, and in two of them he had been able to demonstrate a slight amount of pus. He could do just as well as that in a fortnight with local treatment. Vaccine treatment was cumbersome; there was a period of a fortnight or three weeks before anything was done, and it meant a long time and hard work to reach that stage which, by local treatment, he would guarantee to reach in a fortnight. There was generally no need for vaccine for the cure of general symptoms; the patient's general symptoms would often disappear within a fortnight under local treatment. He desired to know from the bacteriologists something definite as to the length of time during which symptoms remained, when recurring symptoms might be expected, and what steps were needed to combat recurrent symptoms; also he should like to know whether Mr. Goadby would do as Dr. Eyre had done and show some of his cured cases.

Dr. TURNBULL said he had been one of Mr. Goadby's successful cases. His general symptoms were slight, but quite definite. For twelve months he had been suffering from malaise, being always tired, more so in the morning than at night; every morning there was a bad taste in his mouth, his tongue was dirty, and a certain amount of bleeding occurred when cleaning the teeth. He never connected the condition with his mouth, and should have been rather offended if anyone had said he had pyorrhœa. Mr. Goadby, however, found pus along the gum margins and between the teeth, and pus could be squeezed out by pressure on the gums. Treatment was by vaccines and local applications,

and the effect was very rapid and very marked. After the first two injections, which caused no trouble and very slight general symptoms, he felt very much better, and the bad taste disappeared from the mouth after four injections. The treatment lasted for about two and a half months, and by then the malaise had completely gone, and neither that nor the bad taste had recurred in the last eight months. It seemed to him that such a case was certainly a cure if no local condition could now be found; and it was not quite fair, if at the end of two years he developed another pyorrhœa, that it should be said it was a recurrence of the original disease, because it was much more likely there should be reinfection than that the disease should have lain dormant all that time.

Mr. J. F. COLYER said that it seemed to him that dental practitioners were not all in agreement as to what should be regarded as periodontal disease. A certain section would seem to limit it to the slow progressive destruction of the tooth-socket, which is accompanied sometimes in its early stages, and always in its later stages, by the presence of pus. Others, on the other hand, would seem to include acute infective conditions of the gum margin, many of which may be regarded as cases of acute stomatitis. With regard to vaccine treatment, he did not quite take the attitude of Mr. Turner, because he thought it was quite possible that this method of treatment would be found useful in certain cases. He had tried vaccine treatment in eight cases without adopting at the same time local treatment, but he found very little improvement until local treatment was adopted. The point he wished to emphasize was that in considering what effect treatment by vaccines and local treatment combined had had upon a certain case, it was very difficult to say how much was due to the vaccine and how much due to the local treatment, for this reason, that cases, and some of the worst type of cases, would improve in the most remarkable way under local treatment alone. He could not help thinking that where vaccine treatment would do well would be in cases of periodontal disease associated with general symptoms. He could not agree with Mr. Goadby's idea that most of the general troubles suffered by the patients arose by absorption of toxins of organisms from the mouth tissue. He was more inclined to think that the general symptoms were due to the gastro-intestinal disturbances which were started by the swallowing of the pus from the mouth. If the infection were directly from the mouth he should expect to find more reaction in the lymphatic glands, and he did not always find that. In the majority of cases in practice the lymphatic glands did not react as one would expect if there were direct absorption of toxic matter from the mouth. He thought it would be useful if bacteriologists would distinguish very definitely the cases of periodontal disease associated with mouth-breathing and cases that were not. He did not believe that any vaccine, or any local treatment, would ever cure a case of periodontal disease if the patient remained a mouth-breather. Another point, he considered,

66 *Discussion on the Bacteriology of Pyorrhœa Alveolaris*

in discussing the question was what was to be considered a "cure." Was a patient cured who still had in the mouth the potentiality for further disease? The two cases he had seen of Dr. Eyre's he should definitely say were not cured; they both had deep pockets, and, if left alone, the periodontal condition would rapidly get worse. He believed that periodontal disease was mainly a local disease, and that the pus condition was simply due to superadded infection. He believed that if the disease were recognized in its early stage, simple local treatment would cure it without the aid of vaccines.

Dr. CARMALT-JONES said that for about three years he had been trying to treat pyorrhœa alveolaris by vaccines, and had treated something over 20, on the whole, he thought, with fairly satisfactory results: 5 or 6 cures and 8 or 9 distinctly benefited. He had always used the short streptococcus for inoculation purposes. He began to treat with *Streptococcus brevis*, and, on the whole, was satisfied with his results. As Mr. Goadby said, there were considerable difficulties in establishing the precise identity of the various streptococci, *Streptococcus longus* in one culture turning up *brevis* in another. His own impression was that the streptococci were common parasites of the mouth, and in ordinary cases harmless, but under certain circumstances became pathogenic; just as the *Bacillus coli*, which was at most times a perfectly harmless organism, might one day suddenly set up an attack of appendicitis. He believed that the mouth was a septic cavity, and that by vaccines it was possible to get at the streptococci in places that local cleansing would not reach. But having freed the mucous membrane and the adjoining structures, if the mouth were still left septic, reinfection occurred immediately; but, with vigorous local treatment, the removal of loose teeth, and plugging the space between the gum and the teeth, a complete cure might be effected. For about a year and a half he had used a stock vaccine of mixed streptococci from the mouth; it gave good results sufficiently frequently to be justifiable as a method, and it saved the fortnight Mr. Turner had deplored. Other organisms, such as catarhalis or pneumococcus, frequently caused secondary infections, and required special treatment, but he believed that, as a rule, the disease began with infection by the streptococcus of the mouth. He congratulated Dr. Eyre and Mr. Payne on the admirable results they had obtained.

The PRESIDENT (Mr. W. Hern) thought the point Mr. Colyer referred to was a practical one. He would like to have seen some cases treated entirely by vaccine, and not by vaccine plus local treatment. He rather took the view that pyorrhœa was a local condition, to a large extent amenable to local treatment. In order to bring conviction to the mind in regard to the value of the treatment by vaccines, such treatment ought to stand alone for judgment and not be brought forward in combination with local treatment. He hoped that vaccine might come in as a useful agent in the treatment of advanced cases of pyorrhœa where deep pockets had developed and the teeth loosened.

Mr. W. RUSHTON said cases in practice were very often met with in ladies who took the most fastidious care in keeping their teeth perfectly clean. A little gingivitis was noticed, the probe went up, there was a little separation perhaps between the lateral and central, and perhaps a trace of pus exuded on pressure—and that was the beginning of disease in a perfectly clean mouth. If the disease was a dirt disease, he could not understand how such a case was possible. Like Mr. Turner, he had no belief in pyorrhœa being due to mal-occlusion. Some of the cases met with every day in practice had the most perfect occlusion. Whether the fact that people did not masticate with sufficient force to keep the tissues healthy had anything to do with it was another question. He emphasized very strongly the care that should be taken in not giving way to a patient who wished to have teeth preserved that should be extracted. Dentists incurred a great responsibility in attempting to restore teeth in such a condition, for instance, as that described by Mr. Goadby in Class C.

Mr. KENNETH GOADBY, in reply to Mr. J. F. Colyer's unbiased and, he might almost say, reactionary opinion on the treatment by vaccines, said that he could by no means agree with him, or other speakers, in that the swallowing of pus was sufficient to cause symptoms of intoxication, more particularly so as he had, from time to time, found direct infection of the blood-stream had taken place. Mr. Colyer was inclined to regard infection of the lymphatic glands as rare, but he, the speaker, did not consider that swollen lymphatic glands and channels were as uncommon as was often supposed. In reply to Mr. Turner's remarks and those of others who had raised the question of local treatment, he had never suggested that the local treatment was to be avoided or that it was not, in its way, of considerable value; but that it was possible to remove the general toxic effects, or remote chronic infection, by mere local treatment was not in accord with fact. Undoubtedly, if the pouring in of all toxins was cut off, it was left for the defensive powers of the body to remove these effects and those toxins which had already gained access; the vaccine treatment stimulated the defensive powers of the body, to this end in a way which no local treatment could be hoped to do. The same reply applied to the point raised by Mr. Sturridge. No doubt local ionization was an extremely efficient method of treating certain local conditions, but the mistake was always made with regard to the alveolar pyorrhœa, in looking upon it as merely a local disease, and not, as it generally was, a general intoxication due to local absorption. Finally, he could only point out that, if local treatment were of the value that had been claimed for it that evening, there would have been no need to attempt any further treatment for the disease. But, as it was, this disease was regarded as the bugbear of the dental surgeon, and he considered it had been definitely proved that local treatment did not cure the disease; whereas, combined with properly-regulated vaccine treatment undoubtedly cures took

68 *Discussion on the Bacteriology of Pyorrhœa Alveolaris*

place, and he was delighted that the work which he had originated in 1905 had been repeated and confirmed in so many directions.

Dr. EYRE said it was a great many years ago since his late colleague and friend, Dr. Washbourn, stimulated Mr. Goadby to work in the untrodden field of the bacteriology of the mouth. The work that had been done previously was irregular and haphazard, and it was very pleasing to find that the early work carried out under this stimulus had borne such excellent fruit. He himself had felt a very keen interest in the bacteriology of the mouth, and had watched and followed Mr. Goadby's work with a great deal of interest, so that he was not altogether unacquainted with what had been done when he at last found an opportunity to deal with the matter. The cases referred to in the paper were not casual cases. In the dental department of Guy's Hospital there were four dental surgeons and two assistant dental surgeons, who between them saw some 30,000 out-patients per annum, and the express intention of those gentlemen was to pick out the filthiest pyorrhœas they could find and hand them over to the vaccine department to see what could be done with them. It was not surprising, perhaps, that under such circumstances quite a large percentage of the 38 cases had shown general symptoms of a very marked character, and knowing how auto-intoxication was responsible for "rheumatic" pains it was not surprising to find that a large percentage of the cases exhibited such pains. Of the three cases shown that evening one was a recent case, the treatment of which had been commenced since the reading of the paper, and it was not put forward as a cured case by any means, as it was a man who had had twelve or fourteen very bad teeth, and had been under treatment for just about a month. Of the others, one had been under local treatment for six months, during which a large number of teeth had been extracted, and all sorts of heroic local measures adopted, and had been handed over as something the dental surgeon could do nothing with. The third case was one of a very acute osteitis of the jaw, high temperature, and pus welling away from the tooth sockets, the teeth positively falling out; a large amount of necrosis went on subsequently and large pieces of the mandible came away. Neither of these two cases had had any treatment for twelve and fourteen months respectively. The method of working out a subject where two men were associated, one concerned with the clinical side and the other with the purely scientific side, was an excellent one. Each man approached the subject more or less as an expert from his own point of view. Clinically, he himself knew nothing about pyorrhœa, except that the subjects of it were people who had rather fetid breath and a lot of pus round the teeth, and he hardly realized the dental conception of a cure. The absence of pus, the absence of general symptoms, and a general improvement in the patient, seemed to him almost sufficient to justify the expression "cure"; and whether such persons were likely again to become infected and show further signs of disease two or three years hence was a matter he did not intend to

discuss, but, instead, would refer anything relating to the clinical aspect of the cases to his "clinical expert" colleague.

Mr. Goadby had rather deplored that no work was done on the bacilli present in the pus. The reason for that was that he (Dr. Eyre) had had quite a large amount of experience previous to taking up the particular series under discussion, and knew that the bacilli themselves did not offer a very promising field. One of Mr. Goadby's previous papers had shown that the variations in the opsonic index to the bacilli were not sufficiently marked to render it worth while searching for that particular group of bacteria, whereas on the other hand the cocci included many well-known representatives of pathogenic bacteria, and it seemed feasible at first view that they were probably concerned in the infection. Surely, therefore, it was more reasonable to take up a line that promised success rather than to follow the work of the pioneers and simply show that they were correct in not ascribing much importance to the bacilli. With regard to the *Bacillus septus* and the *Bacillus necrodentalis*, the *Bacillus septus* was an organism which undoubtedly did give rise to catarrhal conditions, but that particular organism was not found in any of these extreme cases that came under observation. He could quite believe that in the early catarrhal conditions those bacilli were present along the gum margin, but by the time the other organisms had gained a foothold and had caused the outpouring of foetid pus the *Bacillus septus* had departed elsewhere. The *Micrococcus catarrhalis* group contained a number of individual strains in the same way that the *Bacillus coli* group, for example, contained a number of individual strains. If fifty or sixty different strains of *Staphylococcus aureus* were taken from as many different patients, those strains would be found to differ amongst themselves in a few minor qualities, and could be differentiated, perhaps, into ten or twenty different classes. The same thing occurred with the pathogenic streptococci, and in this connexion he was in the habit of terming a pathogenic streptococcus *Streptococcus pyogenes longus*. The *Micrococcus catarrhalis* was an organism that had sprung into importance during the last two or three years, and therefore had not been worked out as fully as might be. It was customary to say that the true *Micrococci catarrhalis* produced no fermentative reactions when tested on any sugar, but it seemed probable that in a short time that conception would have to be modified and an acknowledgment made that there were types that did produce fermentation on one or two or more sugars at different periods of their existence.

With regard to whether the general symptoms were due to local absorption from the infected tissues of the jaw or to the swallowing of pus, he was inclined to the latter view. In nearly all the cases the gums were indurated and hard, and very little absorption could take place. The effect of swallowing or of absorbing dead bodies of bacteria had been referred to as being a natural process of immunization, much more easily regulated and much better for the patient than the injecting of definite quantities of vaccine under the skin. That might

70 *Discussion on the Bacteriology of Pyorrhœa Alveolaris*

be so, but the fact remained that the local condition remained *in statu quo*. The reason why patients themselves did not get well was that the antibodies present in the blood-serum were not able to get into actual contact with the bacteria owing to the barrier of indurated tissue Nature had placed between the general blood-stream and the pocket of pus containing the different bacteria. In nine-tenths of the cases there was no infection of the blood-stream, but merely symptoms due to absorption of purulent matter. In fact, in the extreme cases already referred to there was no evidence of real blood-infection, with the exception of one, the man with a high temperature and acute osteitis.

Mr. LEWIN PAYNE thought it was not generally understood that the three patients shown that evening had not been ordinary cases of pyorrhœa alveolaris; these cases were specially selected in consequence of their severity. One was that of a patient, under treatment at the present time, who showed satisfactory improvement four weeks after the first injection. The treatment of the other two cases was completed in November or December, 1908. In his experience cases of such severity that had received local treatment alone did not present, after an interval of twelve months, a condition which was comparable to that shown in these two patients.

Odontological Section.

February 28, 1910.

Mr. WILLIAM HERN, President of the Section, in the Chair.

Some Experiments on the Relative Susceptibility of Different Teeth to Dental Caries.

By STANLEY P. MUMMERY, M.R.C.S., L.D.S.

WHEN a little over a year ago I had the privilege of reading before this Section a paper on "Heredity and Dental Disease," I put forward the contention that the varying susceptibility to dental caries found in different mouths could not be accounted for entirely by the varying environmental conditions existing in those mouths.

The theory held by Dr. Sim Wallace and others—that all teeth are susceptible in an equal degree to dental caries, and that its occurrence is determined entirely by the conditions to which the teeth are subjected after eruption, and not in any sense to a variation in the resisting power of the teeth themselves—is difficult to reconcile with the facts of daily observation.

We not infrequently meet with mouths exhibiting all the exciting causes of dental caries, mouths in which quantities of soft carbohydrate food lodge between and around the teeth after every meal and there undergo active fermentation, and yet little or no dental caries results. Other mouths we see where the most scrupulous attention is paid to oral hygiene, and all food particles removed by the brush several times a day, and where nevertheless the most active caries progresses. We all know of cases where the teeth seem literally to tumble to pieces in spite of most careful cleansing and constant visits to the dentist, and this often in mouths where the teeth are perfectly regular and the gums in perfect contact to the teeth. These facts are so well known that the general assumption among dentists is that teeth *do* vary to a marked extent in

72 Mummery: *Susceptibility of Teeth to Dental Caries*

their susceptibility to caries, and various clinical types of "hard" and "soft" teeth are recognized. There are the dense yellow teeth with perfectly smooth even enamel showing a high polish like fine Chinese porcelain, and there are the white, chalky-looking teeth with opaque, lustreless enamel. To what extent, however, it is possible to classify teeth according to their clinical appearances I am not prepared at present to say. The results of my investigations in this direction have not so far produced very definite results, as I shall explain later. The point which I hoped to settle by these experiments was whether the clinical distinction of hard and soft teeth was a correct one—whether, in other words, the teeth showed different powers of resistance to dental caries.

Much work has already been done in this direction, and we are all familiar with the famous experiments of Black in America. This investigator, working on the current assumption that the so-called hardness and softness of teeth were determined by the relative quantity of inorganic salts present, made extensive analyses of teeth to determine this point. By subjecting them to great heat he burnt out all the organic material, the final product of combustion consisting only of inorganic ash. By weighing this and subtracting the weight from that of the whole tooth before combustion, he obtained the weight of the organic material and water burnt off, and so arrived at an accurate estimate of the relative quantities of organic and inorganic components of the tooth. The results were, however, negative, since it was found that the variations in proportion of these two substances bore no relation to the degree in which the teeth were attacked by dental caries. More recently Gassmann¹ published a series of analyses of different teeth made by him, which only further corroborated the result of Black's investigations. In this table he compares the analyses of canines, milk teeth, third molars, senile teeth, and dog's teeth.

	Canines		Milk teeth		Third molars		Aged persons		Dogs
Water	8.09	...	3.76	...	6.91	...	8.17	...	10.97
Loss on calcination									
= organic matter	22.2	...	22.84	...	18.33	...	21.42	...	25.99
Lime salts...	29.78	...	29.59	...	31.65	...	30.25	...	27.33

(*Brit. Dent. Journ.*, February 15, 1909.)

Thus we see that the third human molar, which we know to be very frequently attacked compared with the other teeth, actually contains

¹ *Zeitschr. f. Phys. Chem.*, 1908.

more lime salts than the canine or milk teeth; while the teeth of dogs, which are practically immune to dental caries, contain less lime salts than any of the human teeth. The late Dr. W. D. Miller pointed out some time ago, with regard to the results of Black's experiments, that it was probable the difference in the degree of susceptibility between teeth lies not in the relative proportion of organic and inorganic matter present, but in the degree of chemical combination between the two. Dr. Miller himself carried out a series of experiments upon dentine in this connexion. He exposed sections of human and animal dentine to the action of weak acids, and compared their relative rates of decalcification. He found that, although considerable differences existed in this respect between human dentine and the dentine of certain mammals and fishes, very slight differences obtained between different samples of human dentine. These experiments of Dr. Miller's dealt, as I have said, only with the dentine; had his life been spared he would doubtless have extended his researches to the enamel. This, however, which appears to me the vital part of the subject, he has left to his followers.

The importance of experimenting with the enamel rather than the dentine in an investigation of this nature scarcely needs emphasizing. The enamel is the only part of the tooth which is exposed to the acids of decay in the first instance. It is the first line of defence, and is indeed practically the only defence, since the enamel once disintegrated by the action of acids and the underlying dentine exposed, caries of the latter tissue always follows. No degree of resistance to acids possessed by the dentine can possibly benefit the enamel which covers it and forms a barrier between it and the acids of decay. *It is in the enamel, and the enamel alone, therefore,* that any resisting power against the action of acids must lie; and if any differences really exist between those teeth clinically distinguished as hard and soft in their reaction to the acids of decay, those differences are to be found in the enamel alone. The experiments which I shall now bring before your notice are therefore concerned entirely with the enamel.

In choosing the teeth to be experimented upon, I purposely did not select only sound teeth, since it appeared probable that the sound teeth which I collected would probably be those most resistant to decay. I accordingly included many carious teeth, chiefly with small cavities, so that sufficient enamel surface was present upon which to note the effect of the acid. In my early experiments, I used teeth that had been preserved in formalin, but thinking that the formalin might possibly influence the result, I discarded these experiments and dealt only with

freshly extracted teeth. I had originally intended to conduct the experiments upon the lines of Dr. W. D. Miller's famous investigations on the artificial production of caries, by placing the teeth in flasks containing a mixture of saliva and bread, and incubating at blood heat. I found, however, that this would involve such an enormous expenditure of time in constantly preparing and changing the mixtures that I had to abandon the idea. Moreover it appeared to me that since I did not wish to produce caries of dentine, but only disintegration of the enamel, that I could supply the free acid in weak solution without prejudice to the value of the result. The decalcifying solution I chose was lactic acid of strength 0.075 per cent. This may, and probably does, exceed the degree of acidity usually found in the mouth, but this fact could not affect its relative action upon the different teeth, and it enabled me to make more experiments in the time at my disposal by shortening the duration of each. Moreover when large food masses lodge and ferment between the teeth, I think it likely that the degree of acidity may at least equal this.

My method of procedure was as follows: I first thoroughly cleansed the teeth from all deposits. They were then numbered, and their individual clinical characteristics noted and set down on a chart, their colour, degree of polish, transparency, and evenness of surface. They were then suspended by silken threads in a glass trough containing the acid solution, which was renewed every day. Every twenty-four hours the teeth were taken out and examined with lens and probe. The first signs of acid action on the enamel are loss of polish and milkiness, the latter being the most reliable, as occasionally polish may remain after the enamel has become quite milky throughout. The surface remains quite hard at this stage, and cannot be scratched by a needle. The next stage is chalkiness, when a fine white powder can be scratched off the surface. This is the first sign of commencing disintegration, and constitutes the end of each experiment, since it is obvious that the resistance of the enamel is overcome. An interesting point here was the part of the tooth which usually gave out first. Contrary to what one would expect, the tips of the cusps of molars and bicuspid, and the cutting edges of canines and incisors, invariably show the earliest signs of milkiness and, later, of chalkiness. Why these situations are not the first to be attacked by caries in the mouth is of course easily understood, since they are the positions of all others which are kept the freest from food particles by the tongue and opposing teeth. It was frequently observed that a patch of enamel on the labial or approximal surfaces would show no sign of acid action long after the other parts of the tooth were deeply disintegrated by the acid.

I will describe in detail one experiment in which I purposely choose teeth of marked clinical differences, and will then show the general results of the others in a chart upon the screen.

(1) Canine, dark yellow, very smooth glassy and transparent. Shallow band of caries at gum margin, and small approximal cavity near cutting edge.

(2) Central, white and very opaque in upper two-thirds, lower one-third grey and transparent. Surface irregular and pitted, but with high polish. Small approximal cavity at gum margin.

(3) Bicuspid, yellow ridged and very dull. Polish almost absent ; no caries present.

This experiment extended over four weeks, a most unusual length of time, and I will not therefore weary you with daily details. To summarize the results, the bicuspid No. 3 showed signs of chalkiness after forty-eight hours' exposure to the acid solution, and the whole enamel surface was deeply disintegrated at the end of nine days, except for one small spot which held out a little longer. The central, No. 2, showed slight milkiness after forty-eight hours at one spot and general milkiness after eight days with one chalky spot. The whole surface was disintegrated by the thirteenth day. The canine, No. 1, showed slight loss of polish on the ninth day and slight milkiness on the eleventh day, and not until the twenty-seventh day did any signs of chalkiness appear. This was twenty-six days later than the bicuspid, and seventeen days later than the central. It is a curious coincidence that the only sound tooth of the trio should be the first to fail in such a marked manner. This tooth, however, was extracted a few months after eruption for regulation purposes, and therefore scarcely had time to decay in the mouth. The canine, on the other hand, which resisted the action of the acid for such a long time showed two cavities. It had been extracted, however, from an elderly person, and had only succumbed to caries after many years of exposure to the action of acids in the mouth.

		Days																
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Centrals	...	—	—	1	2	—	1	1	1	—	—	2	1	2	—	1	1	—
Laterals	...	—	1	—	3	6	2	2	1	1	1	1	—	—	—	—	—	—
Canines	...	1	—	2	3	2	1	—	—	2	1	—	—	—	1	—	1	1
Bicusps	...	—	6	8	3	1	2	1	3	1	—	2	—	1	—	—	—	—
Molars	...	—	1	4	7	3	1	3	2	2	1	—	—	—	1	—	1	—
Total	...	1	8	15	18	12	7	7	7	6	3	5	1	3	2	1	3	1
Hypoplastic		1	—	—	1	—	1	—	—	2	1	—	—	—	—	—	1	—

76 Mummery: *Susceptibility of Teeth to Dental Caries*

In this chart I have adopted the natural classification of the teeth, not with any special purpose, but chiefly for the sake of clearness. The table does, it is true, show a slight superiority in resisting power on the part of the incisors and canines over the bicuspid and molars. Such comparison to be of any value, however, should be between equal numbers of each kind, and a far larger number of teeth would need to be experimented with. The hundred teeth recorded here were of all kinds, chosen without discrimination.

The important point brought out clearly in this table is the very marked difference that exists between the teeth in their powers of resistance to the acid. Thus we see as an extreme instance that one canine—in the last column—shows twenty-seven times the resisting power of another—in the first column. The third, fourth, and fifth days appear to represent the average duration of the majority of teeth as seen in the total beneath. Below I have recorded separately the hypoplastic teeth which were included in the experiment. I wish especially to point out the fact that these teeth show a very good average of resistance compared with the rest, as I shall refer to this point again later.

I had originally intended to show a separate chart in which the teeth were classified according to their clinical characteristics, such as colour, polish, transparency, smoothness, &c. In this way I thought it might be possible to determine which qualities were of the most value. When, however, I attempted to draw up the table I found that the results were not worth recording owing to the total number of teeth being quite insufficient for the purpose. Thus it was not sufficient to compare a tooth of high polish with one of poor polish, since other qualities, such as smoothness of surface and transparency, would have to be taken into account. I hope to continue these experiments later, and, by dealing with a much larger number of teeth, to compare different combinations of qualities.

Now, the results of these experiments seem to warrant the conclusion that teeth *do* vary in their resisting powers to the decalcifying action of acids. It further appears to me that if such variation in resistance be proved outside the mouth, it is permissible to conclude that a similar variation would be shown in the mouth. The only difference between the two sets of conditions is in the source of the acid and the temperature, neither of which factors could conceivably alter the results. Of course, outside the mouth it is obvious that all teeth must sooner or later succumb to the action of the acid, while in the mouth teeth

often last throughout a lifetime without showing signs of decalcification—that is, the beginning of dental caries. This is, however, only a question of length of exposure to the acids. Those teeth which in the experiments reacted to the acid in the shortest time would, in the mouth, probably decay in early life, while those which withstood the action of the acid the longest would, I imagine, in many cases, remain free from caries throughout life. In any case, it can only be a matter of time for all teeth to become affected; and here other factors must be taken into account. Thus in clean mouths with regular teeth and healthy gums, where very little opportunity for the lodgment of food occurs, teeth of comparatively low resisting power might remain free from caries quite as long or longer than teeth of a higher power of resistance would do in dirty mouths with spaced teeth offering many crevices for food to collect. At the same time teeth of very low resisting power will give out in a short time in spite of the most favourable conditions; and, again, teeth of very high resisting power will remain sound in spite of constant exposure to fermenting food for very many years.

We have an instructive example of these varying conditions in certain diseases. It is well known that in most febrile diseases of long duration, such as typhoid fever, the teeth are frequently attacked by the most rapid caries, and a previously sound dentition may during such an illness be almost destroyed. This is what one would be led to expect, since during high fever all food retained in the mouth has a greatly increased tendency to ferment, owing partly to the increased temperature and largely to the diminished flow of saliva, which is not only scanty but viscous and acid in reaction, so that, instead of acting as a solvent of retained food particles and helping to wash them away, it rather aids in the fermentative process. Under these conditions the teeth are exposed to the action of the fermentative acids continuously for many days, and even weeks, together. Add to this the usual neglect of oral hygiene which is allowed during severe illnesses, and the result upon teeth even of high resisting power is, I think, not surprising.

Before passing to the last part of my subject I must refer briefly to a theory which has been advanced upon more than one occasion. It has been asserted by several writers that teeth vary in their resisting power to dental caries at different periods of life by the addition or subtraction of lime salts from their composition. It has been suggested, for instance, that during pregnancy the increased tendency to caries often observed is due to the abstraction of lime salts from the mother's teeth to help

to form the bones of the foetus. That this may occur to a limited extent as regards the dentine which is in actual contact with the blood-vessels of the pulp is not impossible, but that the enamel could be thus changed in structure is inconceivable, since this tissue is practically inorganic, and certainly contains no blood or organic fluid which is capable of dissolving the lime salts or carrying them away into the blood-stream. Professor Röse, in the lately published account of his interesting researches into the effects upon the population of the lime salts in their daily food and drink, says that there is a continual metabolic process going on in the body whereby the used-up tissues are renewed. Thus, he says that the old molecules of magnesium and calcium in the teeth—with the exception of the enamel—are continually being replaced by fresh ones, the enamel, however, undergoing no change. The writer especially emphasizes the fact that the enamel undergoes no alteration, the hardness or softness of this structure being determined at the time of calcification during infancy, and there might thus be, he remarks, a very great difference in the resisting power of different teeth to acids due to the differences of calcification in the enamel. I have, therefore, Professor Röse's authority behind me when I say that it is inconceivable that the enamel can change in its power of resistance to the acids of decay after the formation of the teeth. It follows therefore that the enamel can have no recuperative power, and that once affected, in however small a degree, by the action of acids, even if only to the milky stage in my experiments, it is started on the high road to caries. Since starting these experiments I have frequently observed spots of this milkiness on teeth in the mouth, and am watching these to see how soon they will show signs of chalkiness.

What conclusion can we draw from these results so far as they go? It appears to me that we can divide the ætiological factors of dental caries into the usual two classes—viz., predisposing and exciting. The predisposing causes consist of the susceptibility of the teeth; and the exciting causes, the exposure of the teeth to the action of decalcifying acids from whatever source. These exciting causes may be modified in many ways, but since they are invariably the result of the acid fermentation of carbohydrate food around the teeth, they are obviously dependent upon the shape and arrangement of the teeth, and upon the nature of the food which is eaten. The latter part of this subject has already been most adequately dealt with by Dr. Sim Wallace, but the predisposing cause—the susceptibility of the teeth—is still obscure as to its origin.

The question naturally arises, to what agency can these variations in susceptibility found in the enamel be attributed? They may be either acquired or inherited. If acquired, they must be determined during the period of deposition of the enamel by the enamel organ. A plausible theory would be that such variations were the result of nutritional disturbances. It seems not unlikely that malnutrition during the enamel-forming period would result in an imperfectly formed tissue. We have an instance of this in the well-known condition of hypoplasia of the enamel. This affection invariably arises from the cause mentioned, and can frequently be traced to some definite organic disturbance such as scarlet fever, whooping-cough, or measles.

The close connexion between hypoplasia of the enamel and malnutrition in infancy is brought home with especial force in those cases where we find a line of imperfectly formed enamel on the incisors, canines and first molars, at levels corresponding with the exact stage of formation of each at a definite period of growth; and upon inquiry we learn that at that exact age the child had suffered from some definite debilitating condition, such as one of the diseases mentioned.

If, therefore, hypoplastic enamel is to be regarded as a definite sign of malnutrition during the period of its formation, and we seek to attribute to the same cause a low resisting power to acids, then we should expect to find this latter condition present in a marked degree in hypoplastic teeth. As I have already pointed out on the chart, however, this does not occur, the hypoplastic teeth in the experiments showing a very average power of resistance to acids; and I think this fact must militate very strongly against attributing this condition to malnutrition. Apart from nutritional disturbances, it is difficult to conceive of any other conditions which, acting during the period of enamel formation, could modify its chemical structure. We have, as an alternative, the theory that this condition is an inherited one, and the fact that very bad teeth and also very good teeth often run in families; the fact also that certain teeth seem especially liable to decay in all the members of some families, lends support to this view.

As to the manner in which such variations could arise and be transmitted, I have not time to enter into to-night, but possibly some of those present may remember the paper on this subject I read before this Section last year, in which I attempted to show how Dr. Archdall Reid's explanation of the prevalence of general diseases among civilized races, could be equally shown to explain the similar prevalence of dental diseases. Since reading that paper I see that Dr. Archdall Reid's

80 Mummery: *Susceptibility of Teeth to Dental Caries*

work has received emphatic corroboration from that great authority on heredity, Professor J. A. Thomson.

Gentlemen, I thank you for the kind attention you have given me, and can only hope that my humble efforts may contribute, in however small a degree, to the elucidation of the ætiology of dental caries.

DISCUSSION.

The PRESIDENT (Mr. William Hern) said the paper was a most interesting one, and was based on some important and valuable experiments. The question of the susceptibility of teeth to caries was a very fascinating subject. Most dentists had observed that certain varieties of teeth were less prone to caries than others. He had been one of those who thought that colour, whatever underlay this, was one of the determining factors; yellow teeth were less prone to break down under acid action than lighter-coloured ones. According to one of Mr. Mummery's tables, the canine tooth seemed to resist the action of acids longer than any other, and that agreed with what they knew of its behaviour in the mouth, although he had always considered that this immunity to caries had to do more with its shape than with its constitution. He agreed that hypoplastic teeth were less prone to decay than others, especially after they had been worn down and polished.

Mr. F. J. BENNETT congratulated Mr. Mummery on the patience and skill with which he had carried out his experiments. He was the third generation of Mummerys who had devoted himself to anatomy and experimental science, and he only hoped Mr. Mummery would be encouraged to pursue the difficult subject he had taken up. Until his paper had been carefully read and meditated upon, it was hardly possible to express any decided opinion. The line of experiment adopted must be taken in conjunction with analytical experiments, such as Black had carried out. The one and important tissue to investigate, as Mr. Mummery had said, was enamel. He believed Mr. Tomes made his analysis of dentine, and that he did not work upon the enamel at all. The enamel, however, was the one essential tissue to be studied in connexion with caries. With regard to Dr. Röse's observations as to the changes in the tissue, he was reminded of John Hunter's experiment in which he fed pigs on madder, and found that the bones of pigs fed on madder for three months became stained, while another set of pigs fed on madder for three months and then on ordinary food for another three months had no discoloration of the bones at all. But in connexion with the teeth it was found that once the pig had been fed upon madder the teeth were stained with madder for all time, no matter whether the madder food was continued or not. In that way Hunter concluded that the teeth did not change their structure after once being formed.

Dr. SIM WALLACE said the paper appeared to give a rough idea of how much importance should be attached to the question of the resistivity of the enamel to caries. The late Dr. Miller had made some experiments in the artificial production of caries, and said that the thick cusps of the enamel were highly resistant; but Mr. Stanley Mummery's experiments seemed to be in conflict with that idea. He himself had assumed that the enamel in some teeth resisted the action of acid rather longer than other enamel, and consequently he admitted the *rate* of the caries would be influenced by the constitution of the enamel, but it did not affect the carious process at all. For this was a chemico-parasitical process going on at first outside the enamel, and that process could in no way be affected by the quality of the enamel. He did not know that the amount of acid Mr. Mummery had chosen represented what might be considered to be that usually found preceding the formation of a carious cavity in the enamel. The concentration of the acid might go up to '75 instead of '075 per cent., yet he felt inclined to let that pass as representing an average amount, because the tables seemed to show very clearly that the importance of the resistivity of the enamel was practically a negligible thing altogether, as Mr. Mummery indicated that, taking extremes in one case, a tooth might resist for only twenty-four hours, while in another case it resisted for twenty-seven days. Assuming that '075 per cent. of acid was the average amount of acid produced during the chemico-parasitical process in the neighbourhood of enamel about to become carious, then one would expect to find the enamel of the highly-resistant tooth to be in as bad a condition as the enamel of the lowly-resistant tooth twenty-seven days later. So that, at the age of 5, a child with highly-resistant enamel would have as good teeth as a child with non-resistant enamel at the age of 5, minus twenty-seven days; in other words, that the enamel, when it was particularly resistant to caries, retarded the decalcification a few days, or perhaps a month. As far as he could make out, that was the logical outcome of the paper.

Mr. HOWARD MUMMERY did not quite follow Dr. Wallace's remarks because it seemed to him impossible to compare the time occupied in the experiment with the same time in the mouth. In the mouth the acid was only in contact for a short time and was being continually washed away, but in the experiments the acid was continually in contact. The experiments were only intended to show that acid continually in contact with enamel would after a time dissolve it away, but in the mouth the conditions were quite different. The acid in the mouth only acted for a very short time, the alkaline saliva neutralizing the acid. A tooth susceptible to the action of acid might last many years without showing any softening, but it would soften earlier than a tooth that was of a more resistant quality.

Mr. D. GABELL asked for a few more details as to the technique of the operations, whether there was any movement in the acid, and as to the amount of cleansing the teeth received before they were put into the acid. It seemed to him that only in one case had any investigation been made as to whether the

82 Mummery: *Susceptibility of Teeth to Dental Caries*

character of the tooth had not been affected by previous exposure to acid, as the very susceptible teeth might have been teeth that had been exposed to acid before the experiment commenced.

Mr. GEORGE THOMSON said Mr. Mummery had referred to the greater resistance hypoplastic teeth offered to caries. There was practically an early period during the infant's life when the enamel was not being properly deposited, owing to malnutrition, resulting perhaps in an illness, and that was followed by a period of improved nutrition, and an improvement in the condition of the child, so that the enamel would be properly deposited. He thought there must be abundant material for investigation of the teeth of children at an early age when it was remembered that about 108,000 children died annually in England and Wales in the first year of their life. Investigations into infant mortality had shown that 156 children per 1,000 died in the first year. Among the survivors were many who suffered from defective nourishment, and that seemed to point to malnutrition being the chief factor in the relative susceptibility of their teeth; in fact he was more inclined to think the explanation of the phenomena would be found in that direction than in the experiments that had been conducted by Mr. Mummery.

Mr. P. J. PROUD asked whether experiments had been made with teeth that all came out of the same mouth. Probably if teeth were taken from the mouth of a child or a person who had been fed on the lines advocated by Dr. Sim Wallace it might be found that all the teeth were equally susceptible. Comparing one tooth from one mouth with another tooth from another did not appear to be a fair comparative test.

Mr. J. H. BADCOCK asked whether the age-incidence of caries had been taken into account. It might be interesting to compare the age-incidence of caries with the action of lactic acid on the enamel of teeth selected according to the age of the patient. It was possible that the teeth Mr. Mummery had found most susceptible to the action of caries were young teeth, and those found less susceptible old teeth. The point was worth investigation.

The PRESIDENT said the fact that the cusps of molars and canines were the first to fail under the influence of immersion in acid out of the mouth, whereas these positions were so seldom affected by decay in the mouth, emphasized the good result of friction, artificial and natural, on teeth as a preventive measure to caries.

Mr. STANLEY P. MUMMERY, in reply, said he had always been of the opinion himself that yellow teeth were very much harder than blue teeth, and, although his experiments had not borne this out, he had not experimented with a sufficiently large number of teeth to come to any definite conclusion. There were cases where apparently dense-looking yellow teeth decayed very rapidly. In answer to a question by Mr. Betts, he had not dealt with upper and lower teeth separately; it would be certainly desirable to test lower teeth against upper teeth, bicusps against canines, &c., although this would involve years of labour.

He quite agreed with Mr. F. J. Bennett, that the apparent superiority in the canines shown in the table was of no value, because the number of teeth was insufficient. He merely mentioned that the superiority did exist on the chart, although he did not attach any importance to it. Hunter's experiments with madder were very interesting, and he should like to know whether the enamel was stained. If the enamel was stained, it would certainly seem that blood* or some organic fluid capable of carrying the stain could reach the enamel.

Mr. F. J. BENNETT said he was not quite certain, but he believed the enamel was not stained. The dentine was stained. He believed some of the specimens were in the Museum of the Royal College of Surgeons.

Mr. STANLEY MUMMERY said Dr. Wallace's remarks were interesting, and he thought that it was something to have Dr. Wallace conceding the point with regard to the susceptibility of teeth, as he always understood Dr. Wallace was a very strict opponent of the theory that there was any variation at all in the susceptibility of teeth. He thought it very probable that the degree of acidity used was stronger than that occurring in the mouth, but the point was not a very important one. The experiments were not made to find out how long the teeth would last, but merely the relative differences between the teeth in reacting to the acid. With regard to Dr. Wallace's remarks on the time, he thought there was a slight error in logic on Dr. Wallace's part. There were only twenty-seven *days*' difference between the teeth in the experiment, but that showed that the teeth were twenty-seven times more resistant; and supposing that the first tooth had lasted a year, then the other teeth would last twenty-seven years. By making the solution weaker he could have made the experiments last for months or even years. It should be remembered that the teeth were subjected to the acid every minute of the night and day, whereas in the mouth the degree of acidity only reached a high point for a very short period, the food being quickly washed away by the action of the saliva and tongue. With regard to the details of the experiments, he had not attempted to agitate the fluid, although it could be done by means of a rocking machine such as was used for developing photographs. The acid round the teeth might to a certain extent be neutralized by the salts dissolved out of the teeth, and with a rocking apparatus fresh acid would be constantly brought against the teeth. The weakening of the acid by this means, however, would be very slight. He had tested the acid after removing the teeth and had found no sign of such effect. With regard to the cleansing, he brushed the teeth with soap and water, and cleaned off with steel instruments any tartar, being careful not to injure the enamel. Any injury of the enamel was followed immediately by chalkiness after exposure to the acid. In nearly all the teeth that had been extracted by careful examination it was possible to see the bruises caused by the forceps. In his first experiment he noticed that all the teeth seemed to go at two points at once. On examining some teeth he had extracted himself, he found this was due to injury by the point of the forceps at the junction of the enamel and the

cementum, and that particular place went at once. He also washed the teeth in hot water and soda to get rid of any grease that might protect them against the acid. With regard to the probability of the teeth having been exposed to acids in the mouth before they were experimented with, the point made by Mr. Badcock and Mr. Gabell was that the increased susceptibility in the teeth might have been produced by this amount of previous exposure to the acids of the mouth. That point had occurred to him, but the only tooth he actually tested was the bicuspid shown in the second chart. This tooth was extracted from a child eleven years old a few months after eruption, and in forty-eight hours the tooth was chalky, showing a very low power of resistance. The first sign of acid action upon the teeth consisted of milkiness of the enamel and this invariably occurred very early in the experiment. In the case of a tooth showing no chalkiness for ten days, for instance, milkiness was often apparent in forty-eight hours from the commencement of the experiment. If, therefore, the different degrees of resistance shown on the chart were due to the different periods of exposure to the action of acids in the mouth previously, one would certainly find signs of milkiness on many of the teeth before starting. Such teeth, however, as showed signs of milkiness were discarded as unsound, and he did not think for this reason that the differences noted on the chart could be attributed to the cause mentioned. With regard to Mr. Thomson's remarks, he did not think the results of the experiments would have been affected, because supposing the teeth were subjected to a period of poor nutrition such as occurs in the case of hypoplastic teeth, one would expect a low power of resistance to dental caries, if it were due to the same cause. He had examined the actual bands of hypoplasia on the enamel and they showed no increased tendency to decalcification. With regard to teeth taken from the same mouth, he did in one case experiment with such teeth, but did not think it was an important point. He did not wish to test either one tooth against another or the teeth of one person against the teeth of another person; he wished merely to find out whether differences existed in teeth in their rates of decalcification by acids. As far as he remembered in the experiment he did make in that direction, the teeth showed considerable differences, the canines and incisors lasting much longer than the bicuspids and molars.

A Sterilizable Film-holder.

By CHARLES A. CLARK, L.D.S.

A SIMPLE device for holding films in the mouth for X-ray work has long been necessary. There are many ways of holding the film: the patient may hold it in position with the fingers; or, in the case of children, by one of the parents or relations accompanying the child (this is not altogether reliable); or a German-silver plate may be struck up, but this involves time and trouble; wax or stent in a tray may also be used, but this is bulky, and for that reason inconvenient to the patient. One of the best ways, no doubt, is to have the film held by an experienced and reliable assistant, but, owing to the risk of dermatitis or of burning the hand, this course is to be avoided. An ingenious device for holding a film in any mouth was one that was shown to me as being used in one of the German university towns, but it had the serious drawback of being unsterilizable. It was also very bulky.

I have endeavoured to overcome all these difficulties by having a number of holders of different sizes and patterns, some of which I will pass round. Those for the lower premolar or molar region consist of a piece of hard wood, easily penetrated by the rays, about $\frac{1}{2}$ in. wide wedge-shaped. This is attached by screws to a piece of pewter bent at right angles, and at the angle there is a space of about $\frac{1}{8}$ in. In this space is placed the film (previously wrapped in black paper and gutta-percha tissue), and, by pinching the holder a little, it is possible to hold the film quite firmly; but it was found that the holder when bitten upon would sometimes tip up, and so not keep the film close to the gum. To prevent this, I had an extension made so that it should be possible for the patient to bite it on the other side of the mouth and so force it close to the gum. Those for the lower incisor or canine region do not require this extension. The holders for the maxilla have the metal more curved to better fit the palate, and, of course, it is also soft enough to be bent easily for any particular case. For the upper front teeth an extension of metal backwards has to be added owing to the fact that the lower teeth do not come sufficiently forward when biting on an object. It also has the effect of pushing the film close to the gum. These have been very successful, enabling me to obtain some very sharp radiographs.

At first I made these holders of lead, but, as it was impossible to make them look nice, I now use pewter. Of course I am quite aware that they should be made of something which can be nickel-plated, as that is so dear to the instrument-maker; but, if so, they would be rendered unsuitable for these reasons. In the first place, lead is soft enough to be bent to any desirable shape, and, secondly, radiographic plates and films give the best results when they are backed by a metal, because it prevents an electrolytic action from the X-rays injuriously affecting the back of the plate or film. Now the best of all metals for this purpose is lead; and aluminium, which would be much thinner, neater, and lighter, is the worst. Consequently, I now make them of pewter, which looks better and probably would be sufficient to prevent the electrolytic action I have mentioned. So I make no apology for these holders not being of the conventional character.

To sterilize these holders it is only necessary to boil them for not less than half an hour. I have been informed that wood cannot be sterilized, but Mr. Goadby and Dr. Bainbridge are not of this opinion. A test was therefore made up for me by Dr. Bainbridge. One of the holders was kept in a bottle of liquid in which was put some extracted teeth, pieces of wool used to swab out cavities when filling, also swab-bings from the necks of teeth affected by pyorrhœa. After several days the holder was boiled for half an hour and then placed in sterile broth. No streptococci were found, and I take this opportunity of thanking Dr. Bainbridge for making the experiment. Less than half-an-hour's boiling is not sufficient. A quarter of an hour or twenty minutes may be enough for instruments, but then they are conductors of heat and the sepsis is entirely superficial, whereas wood, being a non-conductor of heat and porous, a much longer time for boiling is necessary to allow the heat to penetrate the wood. The thicker the wood the longer the boiling.

A Method of ascertaining the Relative Position of Unerupted Teeth by means of Film Radiographs.

By CHARLES A. CLARK, L.D.S.

THE method I am about to describe is especially useful for fidgety, nervous children, where a stereoscopic view is quite impossible, because it does not matter in the least if the child moves about between the taking of each radiograph. It first occurred to me when radiographing superimposed buried teeth in the incisive and canine region owing to the difficulty of deciding which tooth is in front of the other, both, of course, being penetrated by the rays. If we have two objects, one behind the other, the one at the back is hidden by the one in front. But if we obtain a view from either side, then, of necessity, they both come into view more or less, as the angle at which they are viewed. So that, if we wish to ascertain on which side of the median line a buried canine is lying, three radiographs are taken: the first directly over the suspected tooth, and which we will call the *central* position, and another *mesial* to this position, while the third is taken *distal* of the first or central position. In some cases it is necessary to take a radiograph to first find the tooth and also to get some idea of its location. Each of the three radiographs must be marked immediately they are taken, or confusion will arise. With the three radiographs placed in their relative position, we carefully note the position of each tooth shown in the central radiograph, and by comparing it with the other two the position of the buried tooth, whether it is on the palate or whether it is situated labially, can be ascertained readily with a little practice. It may perhaps be better in some cases to take five radiographs instead of three—viz., one central, and two mesial and two distal, each of which is at a different angle; because, if there are teeth adjacent and crowded, the premolars on one side and incisors on the other, the angle may be too much or too little to guide us.

In the mesial radiograph the supernumerary is seen to be more over the canine, or, putting it another way, the lateral has come more into view; therefore the supernumerary must be situated labially. The distal radiograph shows it more over the lateral than is to be seen in the central radiograph. Through the courtesy of the surgeon for whom

I took this case I am able to show you a model taken subsequently, and you will see that the opinion that the supernumerary is situated labially is confirmed.

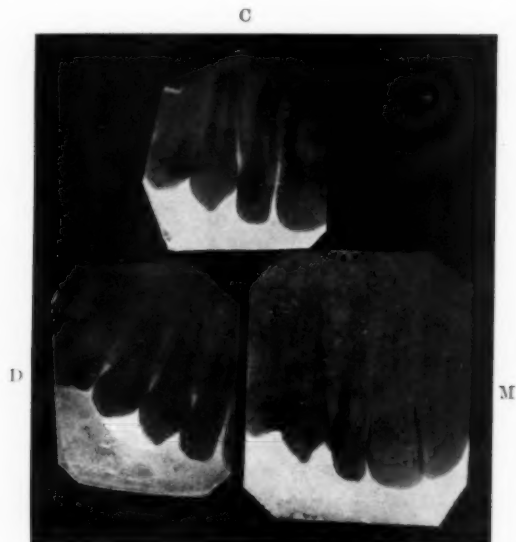


FIG. 1.

Upper left : case of a supernumerary adjoining a lateral. In the central radiograph the supernumerary is seen over the lateral and touching the canine.



FIG. 2.

Lower left : Central radiograph shows the unerupted canine between the lateral and first premolar.

The distal radiograph shows a space between the canine and first premolar; it is therefore situated labially. If this is so, then the mesial radiograph will also show a space between the lateral and canine, which is so. This case shows the advantage of this method, as it was that of a very nervous child who refused to sit still. At last, after much difficulty, I obtained one in the central position; again, after much trouble, the child meanwhile moving about, in the mesial and distal position. As it was impossible to get the child to sit still a moment, the exposure given to each was only a fraction of a second. An exposure of a few seconds only would have resulted in this case in total failure.

DISCUSSION.

The PRESIDENT (Mr. Hern) said the method described by Mr. Clark for ascertaining the relative positions of unerupted teeth was a very ingenious one. Anything that would give such information was of great value in practical work. He had seen something of Dr. Mackenzie Davidson's method of stereoscopic work in discovering the position of shots and bullets, and had been greatly surprised at the results obtained by a good stereoscopic photograph as compared with an ordinary one. If the same results could be obtained in connexion with teeth by Mr. Clark's method, it would be exceedingly valuable.

Mr. DOUGLAS GABELL said the only objection he could see to the method, which he had seen employed at the Royal Dental Hospital, was that it only showed whether a tooth was inside or outside the arch but did not give any distance, and therefore it was not possible to tell how far the tooth was inside or outside.

The PRESIDENT, referring to the film-holders, asked whether they could be turned upside down for the upper or lower, throwing the teeth downwards for the lower and upwards for the upper.

Mr. CLARK said not. He usually got the patients to bite on the top for the lower incisor regions, but for the upper incisor regions it was necessary to have the extension backwards. In the bicuspid region he preferred the extension, as otherwise there was a tilting up.

Mr. RILOT asked whether Mr. Clark trusted to the patient holding the apparatus firmly between the teeth, or whether he tied the jaws together. Some people had a habit of letting the jaws relax at the critical moment.

Mr. BADCOCK asked what the little disk was used for.

Mr. CLARK, in reply, said the method of ascertaining the relative position of teeth was designed really more for fidgety children where stereoscopic views could not be taken. He claimed it was an advantage over the ordinary way of taking a photograph on one flat negative. Taking, for instance, a buried upper canine, it was not possible to tell for certain by the ordinary method whether it was on the palate or situated labially, but the method he had introduced would give that information. Considering that the depth from the labial to the palatal surface was not very great, he thought it practically met all the needs. With regard to Mr. Rilot's question, he relied on the patient biting, and had not found much difficulty in that respect. The little disk was an extension for the bite on the other side of the mouth; it could be bent backwards and forwards for adjustment to the teeth.

Odontological Section.

April 4, 1910.

Mr. WILLIAM HERN, President of the Section, in the Chair.

A Case of Rupture of a Gastric Ulcer while in a Dental Chair.

By W. DE C. PRIDEAUX, L.D.S.

THIS occurred while I was inserting an amalgam in the crown of a lower left bicuspid whose pulp had been extirpated and canal filled some days previously. I mention this to show there was no nervous strain on the patient, whom I had often seen before. The first warning of anything unusual was an urgent motion on the patient's part of a desire to vomit, or, as I then thought, rid herself of saliva while I was disengaging my Ivory matrix; before I had quite finished, a large amount of dark blood was brought up. I gave her about m xv of adrenalin (1 in 1,000), but she brought this up, and the quantity of blood becoming alarming, I sent just opposite for her doctor; meanwhile loosening her dress and tilting chair to the horizontal, for she was rapidly becoming pulseless.

The following notes are from Mr. B. W. Gowring's pen: "I found her in the dental chair; on her clothes and around was a large amount of blood which, from its appearance, had evidently come from the stomach. She was pulseless at the wrist; her face, deathly white, was covered with a cold, clammy sweat; her pupils semi-dilated; her breathing shallow and sighing. To all appearances she was dying. I injected $\frac{1}{50}$ gr. of strychnine hypodermically, warmth was applied to the heart, and, when she had recovered somewhat and was again able to swallow, she was given a teaspoonful of adrenalin chloride (1 in 1,000) with a little

92 Prideaux: *Fracture through Mandible and Canine Tooth*

brandy. This was followed by small lumps of ice by the mouth, and, after some hours in the dental chair, it was found possible to carry her to her home close by in the town. She afterwards had a serious illness; nourishment was administered *per rectum*; the hæmorrhage and faintness being repeated one morning, two days after, whilst she was lying quietly in bed, but not to such an extent as before."

Miss C. is aged 27, and, although she had had dyspeptic symptoms, she had never before brought up blood, nor had gastric ulcer been suspected; her meal on the morning of the occasion related had been a light one of only bread and butter. The patient is now very well and able to perform her duties without difficulty.

Mr. Gowring says: "The circumstances under which she was taken ill must be unique, not only in the experience of dental surgeons, but of doctors also."

I bring it to your notice, having searched in vain for a similar happening. Perhaps we should consider ourselves fortunate, for with nervous patients a weak spot might reasonably declare itself during a dental operation.

A Case of Vertical Fracture through the Mandible and Canine Tooth.

By W. DE C. PRIDEAUX, L.D.S.

THIS fracture happened on board H.M.S. *Dreadnought* while at torpedo practice. Fleet-Surgeon Robert Hill, M.V.O., sent the patient to me, and has kindly given the following particulars of the accident:—

"One torpedo was hung up by a tackle, which gave way and fell on the patient, causing him a lacerated wound on the right temple, the fracture of jaw through the left canine, and severe contusion of the knee. There was scarcely any displacement of the mandible, and what there was was lateral. The tooth was loosened (fractured of course we now know), but seemed to have so much grip that I did not at once remove it. I put a wire through between the lateral incisor and the bicuspid, which appeared to be satisfactory."

The splint had been removed before the patient was seen by me, and the fracture healed with perfect articulation, but the canine remained loose. I found it fractured vertically, and removed the fragments under

eucaine, the lower being particularly firm. It appears difficult to understand how such a tooth-fracture could happen, but looking at the fragments it can be seen how the portions might have been held so that a violent strain could have done it, accompanied perhaps by clenched teeth immediately following the earlier blow.



Mesial surface.



Lingual surface.

DISCUSSION.

The PRESIDENT (Mr. Hern) said the accident narrated in the second communication was an uncommon one, but one which might happen to any dentist. He asked whether the patient had suffered any previous hæmorrhage. The case of vertical fracture was a very unusual one, and it was difficult to understand how the tooth could have fractured longitudinally in that way. He remembered a case in which a very strong lower canine was broken off by the knuckles. One man gave another a blow in the face, which knocked off a strong lower canine with a transverse fracture. Before calling on the reader of the paper, the President said one of the advantages of several branches of the healing art being combined in one Society was that those working in special departments could meet together for the discussion of topics of interest to all. The present was such an occasion, and members of the Clinical Section were specially invited to be present and to join in the discussion of the paper Dr. Ackerley was about to give them.

Mr. PRIDEAUX replied that there was no history of previous hæmorrhages in the case. The accident happened on board the *Dreadnought*; the body which struck the patient was an 18-in. torpedo, which weighed rather more than half a ton. He did not know how the man escaped with his life.

**Observations on the Condition of the Mouth in 1,000
consecutive Cases of Chronic Disease.**

By R. ACKERLEY, M.B.

I HOPE that I shall not be regarded as an intruder in addressing you to-night. Though your Section deals with a branch of medical, or perhaps I should say surgical, work of a highly-specialized kind, there are so many occasions on which the practitioner of general medicine needs your assistance, and in turn on which you need his; there are so many problems relating either to the maintenance of health or the aetiology of disease in which we are jointly concerned, that the more we study these questions together, the more likely we are to obtain useful knowledge. The amalgamation of our various Societies into one Royal Society of Medicine has rendered this joint study more easy, and was one of the main purposes of the amalgamation. So in asking you to allow me to put before you some observations made on a part of the body which is your especial study, I feel I am not departing from, even if I am not initiating, a procedure contemplated when this Society was formed.

Before proceeding to deal with my subject, may I be allowed to say that the title does not really describe it? Those of you who are parents know how hard it is to "name this child," and how often the name that is decided on and given does not suit the child in after-life. It is the same to some extent in giving the title to a paper. I chose the title of my paper as the shortest which would at all indicate what I hoped to deal with, but a better title would be: "The Condition of the Teeth and the Way they are Used in 1,000 Patients with Chronic Illness."

It is now agreed and taught by our profession that for the thorough digestion of food careful mastication is necessary. One can hardly take up a single book on food, dietetics, or general treatment, without seeing some statement to that effect; but when one comes to actual practice one finds that the directions given to patients on the importance of mastication are too frequently given perfunctorily and without conviction. "Eat carefully" seems to be the most that is generally said to a patient, and often even that is omitted. But if careful mastication is necessary for those whom we choose to call healthy, because they are in the vigour of youth or have no marked pathological symptoms, surely

it is much more obviously necessary for those who are failing in health and whose nutrition is imperfect ; especially in the large group of chronic diseases in which there are marked dyspeptic symptoms or faulty metabolism. This includes all the diseases of stomach, bowels, liver, pancreas, and, directly or indirectly, most cardiac complaints; the conditions described as gouty and rheumatic ; and all those in which there is obesity or wasting, or tendency to waste. Quite apart from the definite evils following the swallowing of lumps of food, or imperfectly salivated food, is it not obvious in these cases that it is only by attention to mastication, whatever else we do, that we can hope to improve the impaired nutrition ? A great deal of attention has been bestowed on the dietary of sufferers from chronic complaints, and it is quite common for patients to be supplied with a list of articles of food that they must, or must not, eat ; but very rarely, indeed, do I find that they are told, as I find it is often necessary to tell them, " It is far less important what you eat than how you eat it."

But we may take it that it is agreed that thorough mastication is important. That being the case, one would expect that one of the first things that the medical advisers of people suffering from chronic complaints would, so far as they could, insist on would be attention to the condition of the teeth, the removal of defective teeth, especially if septic, and the correction of any mechanical impediment to mastication. How far this is done can only be found by careful inquiry, and as, with my own patients, I have kept more or less detailed notes of the state of the teeth and how they are used, I thought it would be worth while going through a fair number of cases of chronic illness, selecting a number easy to deal with. The cases are of the type usually seen at the spas ; many of them are of very considerable duration, having been under treatment for many years, often by several doctors, and, on the whole, the patients are genuinely anxious to obtain and follow advice which is given to them with any show of reason fortified by conviction. They are sufferers from various forms of dyspepsia, affections of the stomach and bowels, rheumatism, gout, (so-called) rheumatoid arthritis, arterio-sclerosis, neurasthenia, and the majority of them are past middle life. The information I have got as regards the teeth and habits of mastication is arrived at in the ordinary examination of the patient ; not the minute examination made by the specialist, but, if anything, the minimum that ought to satisfy the conscience of any medical man, however busy he may be when consulted regarding any general condition of health. After ascertaining the kind and quantity of food that is taken during the day,

the question is put, "And how do you treat your food in your mouth?" The answer may be, "Oh, I am very careful to bite it up well," or "I'm rather quick over it"; but it is astonishing to find how often one gets at once the reply, "I know I ought not to, but I am afraid I bolt it." If the admission is merely of quickness, one nearly always finds on further inquiry that this too means "bolting," or at the most that meat receives some slight attention from the molars, but that bread, cake, potatoes, and even fish go down very quickly. But the statement that care is taken cannot be accepted straight away. Very often there is a candid husband, or wife, or daughter present at the interview, who immediately exclaims, "Oh, indeed you don't, you bolt your food like anything; I am always telling you about it"; and, again, a few questions elicit a confession of a rapid meal and bolted food. One man assured me he masticated well with one upper molar and some loose septic stumps in the lower jaw. But even when it is alleged that much time is taken over a meal, this must not be accepted as evidence of careful mastication. A case of a man suffering severely from neurasthenia and colitis with very defective nutrition illustrates this. I was practically sure that one factor in his case was that he bolted his food, but he and his wife assured me that he masticated well, and was always the last to finish a meal. I made an excuse to get him to dine with me alone, and then I found that though it was true that I had to dawdle over each course in order to be decently polite, and even then finished first, every mouthful of his food was bolted. The truth was he spent the dinner-time in talking instead of eating; and if you notice your talkative neighbours at a dinner party you will find that this is a common habit, and makes one doubt the value of gregarious feeding. Having ascertained the method of eating—and may I say that I am careful in making notes not to allow any bias to come in, so that frequently I record statements that food is masticated, though I much doubt it—one goes on to ask, "What kind of teeth have you got?" following this up with, "Let me see them." An answer that they are bad is of course promptly verified, but one that they are good is much too frequently founded on a conception of goodness very different from one's own. One is told they are good when there are no lower molars at all—artificial or natural—or when no two molars meet, and when, though many teeth remain in the head, each one is lying in a bed of pus, or is decayed down to the gum. If one is told that there are few teeth, but that they are supplemented by plates, then one has to see the plates. Often there is an upper plate with no lower teeth, natural or artificial. One ought to

remove and examine the plates, and I have to confess that on some occasions I have not been able to do this, so that I must have failed to record more or fewer cases of plates covering septic stumps, and so far my analysis will be incomplete.

Before giving an analysis of the cases, I wish to say that over 90 per cent. were of really well-to-do people. Not 2 per cent. of them suggested, or would suggest, that want of means prevented them having the teeth put right, and the vast majority of them were going to a doctor or dentist more or less regularly.

In the table I am about to give, I have adopted certain headings. "Excellent" means a mouth containing all the teeth in a good state of preservation, with or without all the wisdom teeth, with no gaps, and with only few stoppings. "Very good" would mean teeth with practically all grinding surface intact, even though many have been stopped. "Good" is applied to a mouth with nothing more than the loss of three molars altogether—i.e. of not more than about 25 per cent. to 30 per cent., or teeth where the gaps, if large, are filled by satisfactory artificial teeth. "Fairly good," if the grinding surface is not diminished by more than 50 per cent. With less than this, I call them "defective." As regards plates, I make no separate entry of a plate, unless it provides at least 50 per cent. of teeth in upper or lower jaw, and I class them as "very good," without comment, or "defective," according to the report of a patient as to the way they can be used for mastication, and their obvious merits or demerits. Loose or imperfect plates are classed as "defective," and I have no doubt that many not so described would by an expert be so described.

And now, taking these classes, what does one find in the 1,000 cases observed: 9 cases, or 0·9 per cent., are described as "excellent"; 22 cases, or 2·2 per cent., are described as "very good"; 198 cases, or 19·8 per cent., are described as "good"; 74 cases, or 7·4 per cent., are described as "fairly good." That is 303, or 30·3 per cent., are either really good or fairly satisfactory, and provide, taking not too high a standard, teeth sufficiently good for mastication.

Now, taking the other classes: Defective mouths number 360, or 36 per cent.; septic—i.e., obviously septic without the minute examination given in a dentist's chair—227, or 22·7 per cent., with 0·8 in addition marked septic with a query. Of these, 185, or 18·5 per cent., were both septic and defective; the rest—i.e., 42—had septic gums with rotten stumps covered by plates. In many of the septic cases there

were smaller plates, but, as explained, I record plates only when they supply 50 per cent. of the teeth.

As regards plates, 87, or 8·7 per cent., are described as good, 250 as plates only, and 20 are recorded as being definitely bad as plates, and are classed also as defective.

Summarizing these, we have therefore: 30 per cent. able to rely on their own teeth; 33·7 per cent. relying on plates, of which only 8·7 per cent. are definitely stated to be good; and 36 per cent. with defective teeth—i.e. with less than 50 per cent. of grinding surface. In the two last classes, 22·7 are obviously septic. Going further, among those not described as septic, there were 20, or 2 per cent., in which foul teeth had only recently been removed.

Now, is not this a rather serious state of affairs, considering the class of patient? Had a hospital or less well-to-do class been taken, I have abundant evidence that the septic and defective sections would be enormously increased. Quite apart from the probability that those who now have plates passed some time with defective and septic mouths, is it not a reproach to us as a profession that more than one in three people in contact with medical men and dentists, and often in contact with men of some note, should be so badly equipped for so necessary a function as mastication? But that more than one in five should possess a foul mouth is still worse. And what makes it worse is that this condition is deliberately allowed by doctor and dentist. Without assuming that all patients tell the truth about the advice given to them, I have direct evidence in not a few cases that even when attention has been directed to the condition of the teeth, a doctor or dentist (and sometimes it is both) has positively advised that a defective or septic mouth should remain in that condition. Let me quote a letter I recently received from a patient whom I saw last summer, suffering from severe dyspepsia, and with a general condition one would describe as severe neurasthenia, which was, I believe, due partly to a want of nourishment, and partly to a poisoning of all the tissues of the body owing to a foul and septic mouth.

February 4, 1910.

Of course, I must not expect any pity from you, as I have not followed your advice and had all my stumps out. I did see my dentist on my return from Llandrindod, and, as neither he nor Dr. X. thought it advisable to have them all removed, I did not have them out. I am having some of them out next week, and if I do not feel better then, I expect I shall have the remainder out, and shall then hope for a speedy recovery.

Now, this letter was written nearly nine months after I had definitely told the patient she could not possibly be well, and I dare to say most positively and dogmatically that it was not possible for her to be well, with a foul and defective mouth.

I know quite well what would be said—viz., that she was not fit for the operations involved. That is—I must be forgiven speaking so positively—one of the most pernicious bits of nonsense talked and acted on that I know of. During the whole of my professional life I have once, and only once, come across a case of *chronic* disease in which delay in extracting teeth has been necessary for more than a week or too, and then the mouth was carefully attended to, to render it less septic, and finely divided food was administered to give the digestive organs a chance.

This was a case of a man, aged 35, suffering from what would at one time have been called pernicious anæmia, but now is more correctly designated "septic anæmia." He was very ill, and had been going downhill for over two years. During the whole of that time he had been under medical advice, and had been visited once or twice a week. The mouth was markedly affected. The teeth were carious and the gums inflamed and septic. Under suitable treatment, beginning with a proper cleansing of the mouth, and later an extraction of the offending teeth, the man made a good recovery. His regular medical adviser informed me that he had been waiting for the patient to get better before he had his mouth attended to!

But to wait week after week, month after month, I may almost say year after year, to get a patient into a condition to attend to the *fons et origo* of his illness, expecting, I suppose, that something wonderful in the therapeutic line will turn up, is, I venture to suggest, foolish, and will some day be regarded as professionally criminal. For over sixteen years it has been my practice to deal instantly and without compromise with these cases—a large number of them with the assistance of my friend Mr. Mellersh—and never once have I regretted, and never once has a patient regretted, the prompt treatment, as the improvement in health has followed immediately on whatever operative procedure was necessary.

Let me give another and even worse case: Eighteen months ago I saw a well-to-do patient in a very serious condition of health, crippled with what was called rheumatoid arthritis, a great sufferer herself and an anxiety to others. One obvious factor was a septic and defective mouth. That is, in a case of markedly defective nutrition, food could not only

not be properly prepared for gastric digestion, but, to make things worse, was fouled and poisoned by a purulent secretion which was also passing into the stomach by day and night. Had I had a dentist at hand, I am sure I could have got her mouth attended to there and then, but I had no one within many miles. She went to town, and then passed under the care of a doctor who had attended to her for some time. He regarded her as unfit to have her mouth attended to, and so for another year she went on, until late last autumn she came under Mr. Mellersh's care in a condition no better fitted for interference with the mouth than she was twelve months before. I describe the condition in which he found her in his own words: "Teeth large and well developed. For this patient it was necessary to remove the roots of the upper left third molar, the first and second premolars, and the second right premolar, these having chronic abscesses discharging from fistulous openings in the gum. Gold caps had been applied some years previously to the following: Upper right, second and third molars, upper left, first and second molars, lower right second premolar, lower left second premolar.

The gum having receded, caries had attacked the roots and crowns of all teeth, with the exception of the upper left first molar, and the lower right second premolar. The caps had been badly fitted and there were spaces between the natural crowns and the encircling metal. Presumably at first the spaces were filled with cement, but most of this had disappeared, and there was a considerable amount of decomposing material under the caps. It was therefore decided to remove these metal crowns, and it was found that all the teeth were extensively carious with the exception of the lower right second premolar. The carious matter being removed from the teeth, it was discovered that the only ones worth saving were the upper right third molar, the pulp of which was exposed, and the upper left second molar. This latter was dead, and no attempt had been made to treat the roots; the pulp chamber contained a mass of septic wool covered with cement. The incisors and canines presented numerous cavities, some of which were filled with gutta-percha in a more or less septic condition."

Now, what plea can possibly be put forward to justify a condition like this being allowed to exist month after month, even year after year, with a patient going slowly downhill? This is again only an illustration; such cases are common. But it is to be admitted that it is usually in cases where there are fewer foci of sepsis that the dentist and doctor are complaisant. But surely organic septic matter does not

become harmless, even if the quantity swallowed is relatively small. Emphasizing the words differently, perhaps, from what Tennyson intended, may we not say:—

“ It is the *little* rift within the lute
That by and by will make the music mute.”

I feel convinced myself, after holding a post as medical officer to an isolation hospital for many years, that the severity of throat symptoms in diphtheria and scarlet fever, and of the severity of an attack of enteric fever, depends largely on the presence or absence of oral sepsis; and in such a disease as appendicitis there is much reason to believe that oral sepsis has much to do with the more severe cases in which a catarrhal rapidly becomes a purulent inflammation. The extraordinarily rapid improvement in the dyspepsias and other troubles of people where there is only slight sepsis when this is attended to by itself justifies such attention and makes delay blameworthy. The importance of a clean mouth to health and longevity is being more and more—though too slowly—recognized by the medical officers to insurance companies. The following extracts from addresses by Sir James Barr and Dr. Hector Mackenzie are interesting and instructive:—

SIR JAMES BARR.¹—A foul-mouthed individual is liable to many troubles, and he is a greater danger to himself than others. The nose, mouth, and bad teeth afford a large culture field for the growth of many pathogenic organisms which are ever ready to attack the individual when his resisting powers are lowered. In the mouth and throat the organisms are bred which give rise to such fatal diseases as pneumonia, pernicious and other septic anæmias, &c. If the insurance companies added to the premiums $\frac{1}{2}$ per cent. for every bad tooth in the applicant's head they would do some good to the dentists and lessen their own risks. The great increase in recent years of diseases of the digestive tract, such as cancer of the stomach and large bowel, appendicitis, &c., are largely due to dietetic errors and constipation.

HECTOR W. G. MACKENZIE.²—I need not enlarge on the importance to health of the body of sound teeth and gums. In many of the medical report forms the examiner is specially asked to report on the condition of the teeth and gums. Unhealthy or defective teeth are responsible for much chronic dyspepsia and consequent ill-health, but there is one disease of great prevalence and of vast importance affecting the gums primarily and the teeth secondarily, which at the present time is ill understood by many members of our profession

¹ *Brit. Med. Journ.*, 1908, i, p. 243.

² *Lancet*, 1910, i, p. 702.

—Rigg's disease. You find the margin of the gum red and swollen and pus exuding from the sockets of the teeth. A slight degree of pyorrhœa may be passed over, but a high degree is incompatible with a good condition of the general health, and must always be considered as a source of danger. In my opinion no life with a decided pyorrhœa is a good risk, and acceptance should be postponed until the mouth has been put in a healthy condition. When I come across a pale, unhealthy-looking subject, I always suspect among other things this disease. When a proponent who wears artificial teeth is under examination he should be asked to remove them. I have frequently found that underneath the plates there are stumps with pus oozing from the sockets. Many a case of obscure health is due to a septic condition of the mouth, a condition which, because unsuspected, is not looked for and not discovered.

I am in agreement with both, but think Sir James Barr's suggestions go further and are more practical than Dr. Mackenzie's. We want the slight sepsis to be recognized and dealt with drastically. A small destructive fire is potentially as dangerous as a big one.

It is true that both doctor and dentist have trouble to get patients to consent to having gums cleaned and septic roots removed, as it is not always possible to make them believe that their suffering is due to the condition of the mouth. But I find that one way of putting it appeals to them. They readily agree that they do not care to eat dirty food or food on a dirty plate. Without using long words, one then tries to show them that they are themselves soiling their food. Another argument is, they would not suck and swallow the pus from a suppurating wound on the hand. Then why swallow pus arising in the mouth?

Now, in all these cases it may be said that even admitting that a clean mouth and sufficient grinding surfaces are necessary, except in the cases of chronic dyspepsia there is nothing to draw attention to the alimentary canal. This is not so, as dyspeptic symptoms are marked in the majority of cases. Of my 1,000 cases no less than 529 complained of, and were continually treating themselves or being treated for, constipation; 57 had chronic or intermittent diarrhœa, and 576 flatulence. So that there was obviously something wrong with the food passage.

This brings me to the habits of these patients, toothful or toothless, in treating their food: 507 confessed that they bolted their food, and to that number I add 91, who had teeth so defective that even moderately good mastication was impossible, making 598; and 798 confessed that when soft food was taken they let it go down without any mastication or insalivation, using their mouths like the opening into a letter-box. I use the expression "posting" food for

these people, borrowing the expression from a writer in one of the medical journals several months ago.

But though it is impossible to enter into the necessary details in a short paper, I have found an enormous difference in the vitality of those who with a clean mouth bolt their food, and those who necessarily bolt it because of defective mouths; and again there is the class with the worst general health, the 22 per cent. with septic mouths.

Now, whose business is it to see that our patients learn to keep their teeth in working order and their mouths clean? Of both doctor and dentist, I think; but the dentist has special opportunity of impressing the teaching on his patients during examinations, and in carrying out mechanical work he has time and opportunity to tell his patients how to use the teeth and the importance of using them; he can dwell on the loathsomeness and danger of a foul mouth: he can tell them the results which necessarily follow bolting or "posting" food. One may regard, too, the care and use of teeth as being especially in his department of medical work. In a large class of patients—the young—I understand that careful mastication is important, if not essential, in producing a good set of secondary teeth. But are mothers and the children themselves sufficiently impressed with this necessity? Get the child to acquire good habits and no one can estimate what has been achieved in the best work our profession can do—viz., in the prevention of disease. As regards "posting," not sufficient has been said and taught. But is it not obvious that the food which is softened and rendered pappy or semi-liquid by the combined assistance of miller, cook, and nurse, is nearly always farinaceous—the very food which requires most careful insalivation for its digestion?

You who are here doubtless do preach and teach as I suggest, but an enormous number of men do not, and so I urge on you that you should not only teach your own patients yourselves, but, in season and out of season, impress on your fellow practitioners and students the desirability of teaching their patients. And the teaching should be with conviction, so that it may be impossible for patients to say, as I often hear them say, "Oh yes, I've been told I ought to masticate properly, but not in a way to make me think it was really important."

DISCUSSION.

The PRESIDENT (Mr. Hern) thanked the author for his extremely valuable and lucid paper, on which he anticipated an interesting discussion. He thought he could assure Dr. Ackerley that he was, to a large extent, preaching to the converted in that Section. Still, the paper dealt with matters which required a great deal of reiteration.

Dr. MACNAUGHTON-JONES said he would not allude to the consequences of want of mastication and deficient salivation. These helped surgery in the direction of "gastro-enterostomies" and "short-circuiting." It was from the surgical view of the interesting paper they had heard he wished to speak. As far back as 1890 a communication of his on "Dental Reflexes" had appeared in the *Dental Record*.¹ In it he urged that "the source of a distant neurosis (arising from the teeth) was hardly kept in view as frequently as it should be in the daily practice of the practitioner and the dental surgeon," also that in carious and otherwise affected teeth we found an explanation of some puzzling condition which had baffled the therapeutic skill of the physician. In 1904 he brought the subject of attention to the mouth and teeth before and after pelvic operations before a meeting of the British Gynaecological Society, urging that in such affections as parotitis and angina Ludovici direct infection by toxins from the teeth, rather than the neural theory, or immigration of organisms from distant organs in the pelvis, explained these occasional post-operative conditions. The anatomical relations and communications of the fifth nerve with the sympathetic, and the lymphatic distribution between the parotid, submaxillary, internal maxillary, sublingual and cervical glands sufficiently explained both the morbid reflexes and the septic invasion. Parotitis, however, was only one of the evils that follow from unhealthy conditions of the mouth after operations. He had then and subsequently urged the great importance of attention to the mouth and teeth before and after pelvic operations. A septic mouth might produce gastric complications and post-operative intestinal sepsis. It was his invariable practice in all cases in which the mouth was unpleasant to make the patient use an antiseptic mouth-wash frequently; say every hour during the day, and it was remarkable what relief it gave. He had advocated the use of cyllin, both locally as a wash, and internally as a preventive of sepsis. He considered it, as its co-efficient was ten times greater than carbolic acid and fifteen times greater than formalin, to be the most powerful of our germicides. Formolyptol and glycothymoline, with peroxide of hydrogen, made pleasant bases for it. One word he should like to add with regard to plates. It was certain that people were far too careless in the wearing of these; they were not alive to the dangers of dirty plates. He had many times seen them worn habitually over foul, decayed stumps, and in some instances the plates were so fixed that it was difficult to remove them. He would like here to say how much the medical profession was indebted to dentistry for the assistance it received from dental surgeons in the treatment of various diseases.

¹ *Dental Record*, Lond., 1890, x, p. 337.

Mr. C. EDWARD WALLIS said he had had considerable opportunities in the last few years of watching the effects of oral sepsis in London County Council school children. In certain schools there were dental charts showing the weights and the average ages of the children; and these clearly showed that the children with the most septic mouths were not only below the average weight of their class, but were below the average intellectual status of their age. Those with the most highly septic mouths were frequently two standards below what they ought to be in accordance with their age. With regard to oral sepsis, it had been his plan at the hospital to have the patients weighed weekly after wholesale extractions of teeth; and, in spite of their being left without being able to masticate their food, they progressively increased in weight, and at the end of a few months they had in nearly all cases gained several pounds. That seemed to show that the really serious matter was not so much the want of mastication as the fact that they were constantly swallowing the products of decomposition and the micro-organisms of disease. With regard to the future, one of the most serious charges which had been made by the lecturer was the apathy of the medical profession in the matter, and that was due, he thought, in many instances, to ignorance. In days gone by there were not "teaching" dental departments attached to large general hospitals; and carious teeth were seldom recognized as needing treatment unless they had large cavities in them. But now in many London hospitals students were instructed in the various points with regard to the teeth, especially in regard to the recognition of pyorrhœa alveolaris. They were now in a position to detect the slight as well as the advanced cases, and also to realize the seriousness of the condition in its relation to the general health of the sufferer.

Mr. HUNT said that as a dentist of many years' experience he had been delighted to hear the present evidence from a member of the medical profession, as it showed that that profession was waking up to the beauty of the gospel which dentists had been preaching for so many years, though often to deaf ears. Dr. Macnaughton-Jones had shown how much internal disease might spring from bad teeth or an unhealthy condition of the mouth. In that matter it was well to remember that a man might have 50 per cent. of good teeth and yet not be able to masticate anything, for one jaw might be quite edentulous. The great point was, what teeth *met*, and *how efficient* these were. Again, any disease which made a single tooth tender, crippled the whole of that side of the mouth; and that was much more the case when there were half a dozen teeth affected. He had often asked patients who came with septic mouths how long they expected to go on swallowing such material from gums and teeth charged with all sorts of abominable microbes without suffering internally. He was very glad to see that medical men were waking up to the value of a healthy mouth.

Mr. J. G. TURNER said he did not think such an analysis as Dr. Ackerley's had been attempted before, and the author deserved thanks for his endeavour. He wished to ask Dr. Ackerley whether, in reviewing those cases, he could make

any deduction as to which was the more important—the sepsis, or the loss of masticatory power. He had long agreed with the view expressed by Mr. Wallis that sepsis was the all-important matter. The loss of mastication was not important until the question of the teeth interfering with each other or with the gums came into the discussion, and then the patient was better without any teeth at all. He asked whether Dr. Ackerley had found perfectly edentulous people—without their own or artificial teeth—with chronic disease, getting on well. His impression was that they would do well. He had seen cases which had been diagnosed as Addisonian anæmia, and apparently correctly, going on very well after removal of the teeth; and in other cases where one would say there was no specially established chronic disease the patients got on well without teeth. He also asked whether Dr. Ackerley had made a note of the form of artificial substitute which had been used by his patients, and, if so, what he regarded as unfit substitutes. He (Mr. Turner) considered that crowns, and especially ill-fitting gold caps, were the most injurious. A gold cap should practically never be allowed. Perhaps Dr. Ackerley could give some statistics from his thousand cases. The author said there was constipation in 500 of his cases, and it would be interesting if Dr. Ackerley could give any sequence of events leading to that constipation. What had exercised his mind had been, what was the smallest degree of sepsis which it was justifiable to regard with suspicion? The only answer he could give to that was that the patient's resistance was the measure of the danger; and resistance was an unknown quantity until it had been tried. Perhaps, with the assistance of such analyses as Dr. Ackerley had made, one might be able at some future date to know where the limit of safety might lie as a general rule. Also, had Dr. Ackerley found it possible to isolate any guiding symptom among the general clinical symptoms of the patient which would lead one to say it was a case of sepsis from the mouth, rather than of sepsis from any other part of the body?

Dr. W. J. MIDELTON said he had listened with much interest to the paper, and he agreed with practically all that had been said. For many years he had paid particular attention to rheumatoid arthritis, so-called, though he preferred to name it arthritis deformans. The teeth played a large part in the causation of that condition; he had treated some of those patients for a certain time without the teeth being attended to so as to test the point; by the method he employed he was able to get the arthritis to recede considerably without attention to the mouth; indeed, patients were sometimes so feeble that he did not suggest any interference with the mouth at first. People often seemed to resent any suggestion that the teeth or mouth should be attended to or kept clean; and that may have led to some extent to the condition being shirked by medical men. He had often raised the question before medical societies as to the rôle played by pyogenic organisms, as shown by clinical observation. He had concluded that pyogenic organisms were beneficent in that they helped to kill off the specific organisms, such as those of typhoid fever, pneumonia, or influenza. In his present method of treatment he regarded pyogenic microbes

as his allies rather than as his enemies. He agreed with those who said that getting rid of the poisoning of the tissues was the important thing. One young lady came suffering from arthritis deformans and exophthalmic goitre; she had previously had treatment under the best circumstances, including the Weir-Mitchell treatment, under which latter she gained only half a pound in weight. He employed an advanced form of counter-irritation, and she was now three stone heavier than when he first took her in hand. He told her her teeth were not in very good order, but she replied that they had had so much attention paid to them that she was now sick of the subject. It was suggested that as there was tartar round the gums it would be well to remove one or two teeth at least. Eventually he persuaded her to have them all extracted. A week afterwards she said she felt very much as she did before she began to be ill; that something had happened, and that she was going to get well. There was a very marked change in the patient. The symptoms of hypersecretion of the thyroid gland had disappeared and her arthritis was rapidly subsiding.

Dr. JAMES GALLOWAY said that he should feel very ungrateful, as a member of the Clinical Section, if he did not offer his thanks to the Chairman and Council for giving him the opportunity of hearing Dr. Ackerley's paper. Coming from Charing Cross Hospital, he would be still more ungrateful if he did not recognize the constant stimulus given to the study of the care of the mouth and teeth afforded by being surrounded by colleagues much interested in this subject. He could assure Dr. Ackerley that if he considered in speaking before the Odontological Section he was preaching to a convinced audience, those present engaged in other fields of medical work were equally convinced of the importance of the subjects to which he alluded. Part of his duties at the hospital consisted in conducting the physical examination of candidates for the nursing staff, and it had been for years his custom in this examination to pay special attention to the condition of the teeth. It was startling to find how many young women coming from educated classes of society had to be rejected owing to unsound teeth. He fully appreciated the efforts of dentists to educate doctors in this matter, but there were instances in which the instruction came from the other side. He remembered a young lady coming armed with not only a certificate from a doctor but also from her dentist stating that she was a fit person physically to become a probationer-nurse. On making the usual examination he found that she did not come up to the standard so far as the teeth were concerned, either as to the number of teeth or the condition of the gums and mouth. He felt that, especially in cases of surgical operation, it was unnecessary to have nurses in charge presenting the opportunities of avoidable septic infection. He thought that the surgeon who was performing an aseptic operation, and had such a nurse to attend on the patient, would naturally have some serious criticism to offer concerning the physician who had passed her fit for service. After he had referred this young lady for treatment he had a letter of almost abusive character from the dental surgeon, and he mentioned this case by way of presenting the other side of the picture. He wished to enter a

protest against what seemed to be becoming the common practice in almost all cases of pyorrhœa alveolaris—namely, wholesale removal of the teeth. He recognised the energy of his dental colleagues present, and trusted that a portion of it might be diverted to efforts for the prevention and amelioration of this state of affairs rather than to reduce the sufferer to the edentulous condition. Mr. Turner seemed to regard this unhappy state as inevitably the eventual condition of those affected; he would protest, however, against the beautiful picture of the toothless condition as drawn by Mr. Turner. Mr. Turner would recollect that this state had already been described by a great authority. We were told that the sufferer was, "*Sans teeth, sans eyes, sans taste, sans everything,*" but it was the feature of the "last scene of all," and not that of vigorous middle life. A case occurred to him, by way of illustration, in a man aged about forty years, who was engaged in vigorous work. He had a strong set of teeth, but unfortunately much pyorrhœa. He was condemned to have his teeth out, but refused, and no one could fail to sympathize with his refusal. He might have gained in weight, as Mr. Turner remarked, but it was difficult to see what benefit would accrue. He would certainly have been refused work on all sides on account of his edentulous state, and the increased appetite for food would make his condition only the harder. He was glad to say that by perseverance, and by getting the patient to understand the condition and to carry out treatment, the state of the teeth and gums had greatly improved; he was not edentulous, he was in full work, he had gained weight in spite of not losing his teeth, and he (Dr. Galloway) was afraid there was no chance of persuading him to have them removed now. Dr. Galloway said that, in some cases there seemed to be a tendency to put the cart before the horse—e.g., if patients suffered from some chronic disease, and also showed a slight amount of pyorrhœa or other septic condition of the gums; an exacerbation of the chronic ailment might occur, followed by severe aggravation of the septic condition of the mouth, but under those circumstances the probable explanation was that the mouth condition, just as any other septic state, grew much worse when for any other reasons the patient's health was lowered. Dr. Galloway said that, though he had ventured to make these criticisms, he entirely agreed with the strenuous efforts required to be made to convince the public of the necessity for a much higher standard of mouth cleanliness than is usual, and especially the prevention and cure of septic oral complications.

Mr. PETER DANIEL said that every part of the human body was liable to be affected by sepsis in any part of that body, so that the dentist was doing a service to the specialist in every department by making the mouth healthy. From his point of view as a pathologist, he quite agreed with what Mr. Turner had said about crowns to teeth; he did not see how one could place a foreign body in connexion with any surface tissue—i.e., epithelium—without causing suppuration, whether that substance were gold or anything else. The gum must be attached by its free margin to the tooth membrane, and there must be a minimum of sulcus between the gum and tooth. One could attach a gold crown

to the remains of a tooth which was raised above the gum, and if none of the gold were sunk below the level of the gum the latter would remain healthy; and that led him to refer to clasps, in regard to the value of which there was much difference of opinion. Every clasp attached to a plate meant the ultimate sacrifice of the tooth around which it was placed. If it is possible, as it certainly is, to make a plate without a clasp, that is the form for use. With regard to antiseptics, cyllin was good for washing the floor; but he did not think cyllin or any other antiseptic would cure pyorrhœa or do much good to the mouth unless the cause of the sepsis were removed. One could not sterilize the mouth, as had been shown, in Germany especially, where such matters were elaborately investigated. The only mouth washes which were really good were those containing alcohol, and these did most good in pyorrhœa in connexion with Bier's passive congestion. He believed antiseptic lotions did good in the mouth merely mechanically, in the same way as washing the floor did good. Astringents were good, and as the power of the medical man consisted in increasing the resistance of the body, an astringent lotion did much good by constricting the blood-vessels and lymphatics—i.e., toning up the gums. He did not think pyorrhœa could be cured by anything short of extraction. Mr. Turner's questions were most pregnant ones. It seemed that only a few enlightened dentists seemed to realize what sepsis meant. One speaker suggested that pyogenic bacteria could be utilized to combat other bacteria; but he (Mr. Daniel) would not like to submit to that treatment. One could afford to ignore the lactic-acid bacillus. He paid much attention to intestinal surgery, and he could not conceive any good from the use of pyogenic bacteria; indeed, they would act in preparing the ground for typhoid and other specific bacteria, and, when those bacteria were present, would encourage sloughing, hæmorrhage, and perforation. All germs in the body are injurious. He regarded vaccine treatment as on a par with treatment by "Bile beans" and did not think one patient would be the worse by the disappearance of vaccine therapy from the physician's armamentarium; he said that after trying it and carefully observing its effects. Take the case of a patient with carbuncle. Every day there must be death of some of the causative bacteria, and these would be taken up and would circulate in the lymph and blood stream; any endotoxins, anti-toxins, &c., which were produced in a test tube by cultivation of these bacteria would be engendered and utilized in the patient's body—i.e., auto-vaccination must constantly be taking place, certainly in these minor ailments, and by vaccine therapy nothing was put into the body which the patient did not already possess. Vaccination in such cases did harm rather than good. On the same principle, pyorrhœa could not be cured by vaccines, and more especially as, once initiated, the condition was largely maintained by mechanical conditions, quite apart from vital questions. What was the standard of cleanliness as to the teeth? What one dentist called clean another did not. When dentists came to a decision among themselves on that point, they could throw the onus on the medical man. Physiological experimental work showed that whether meat was swallowed as a bolus, or finely divided, the

110 Ackerley: *Condition of the Mouth in Chronic Disease*

time for digestion as evidenced *in vitro* was but little altered, so that the removal of sepsis was immensely more important than the presence of masticatory power. Another important matter was the relationship of diabetes to oral sepsis. A septic urethra or septic skull was not the same thing to a patient as a septic intestinal canal; the latter must be of the greatest potency in producing glycosuria, which everyone agreed was due to pancreatic disease. Since 1900 he had seen 22 cases of complications of diabetes which had come into his hands as a surgeon, and he had never seen a clean-mouthed man or woman among them. In his opinion this disease occurred most frequently in the people who most neglected the hygiene of the mouth. In seeking for oral sepsis, the majority absolutely ignored the gums, crowns, and clasps of dentures, such data being really indispensable to any consideration of oral sepsis.

Mr. STURRIDGE said he had heard that evening three of the most remarkable statements that he had listened to in this Society. It was strange to hear a medical man say pyorrhœa should be treated and could be cured, when more than 75 per cent. of the men practising dentistry in England said it could not be cured and did not try to cure it. He did not think there were many cases of pyorrhœa in which the dental condition could not be much improved by treatment, and conservative measures should be tried before resorting to extraction. Another medical man said that dentists did not recognize tartar as a condition of the teeth which called for interference. That was a fact, and accounted for so many cases going on to pyorrhœa without cure. A third remarkable statement was the advocacy of the non-use of clasps. It was reasonable that any tooth which was clasped would decay in time. He had often cut off clasps from other men's plates, and the patient got on as well without. It was a matter of capillary attraction and proper fit.

Mr. LEWIN PAYNE said there had been various contradictory statements made in the course of the discussion, and it was one's duty to give one's views on certain points. He strongly opposed the statement of Mr. Daniel about the vaccine treatment of pyorrhœa alveolaris, and he was sure cures were obtained by that treatment in cases which did not yield to any of the other methods at present employed.

Mr. SIDNEY SPOKES said the present-day treatment of setting a microbic thief to catch a thief seemed to have another bearing than that which had been only casually alluded to, but was possibly destined to have an important effect. Those who fed a patient on lactic acid provided the patient with something which would help to destroy his teeth unless precautions were taken. He recently saw a very suggestive case in a young lady who until now had been fairly free from caries. She was now taking lactic-acid milk twice daily, and without the precaution of washing her teeth. She had half a dozen cavities in

her teeth, the entrances to which were only small, but a considerable part of the substance of the teeth was involved. He was on the side of those who advocated wholesale extraction whenever that seemed justifiable. A few years ago he published records of cases selected from physicians' cases in the wards of University College Hospital to show how many instances of anæmia and suspected malignant disease cleared up after removal of the diseased teeth. He recently saw a woman who was admitted with the suspicion that she had typhoid fever; but the temperature oscillated in a remarkable way, and streptococci were found in the blood. She had pyorrhœa in all the sockets of both upper and lower jaw, and immediately after removal of the teeth she began to get better.

Dr. ACKERLEY, in reply, said one sometimes brought forward a discussion because one wanted encouragement. He often felt he was carrying on a fight single-handed against both doctor and dentist; but he had had more fights with the dentist than with the doctor, especially with regard to slight oral sepsis. He did not recognize that a small fire was ever safe, as at any moment it might become a big one. One could not imagine the London Fire Brigade refusing to turn out to a fire because it was only a small one; and definite sepsis should not be allowed to exist in the mouth without attempting to get rid of it. His invariable experience had been that the health was improved when the mouth was cleaned properly. He did not think sepsis and the lack of proper mastication were in the same category. Still, he did not regard the bolting of food as at all safe. Carnivorous animals could and did bolt their food and do well, but he believed no herbivorous animal bolted its food, and man was largely herbivorous. Carbohydrates especially should be properly insalivated. He had notes of a considerable number of cases who suffered from chronic constipation, or flatulence, or diarrhœa, who had not septic mouths, but who did not take care to properly insalivate their food. By properly masticating food, constipation could in many cases be got rid of, though perhaps not for weeks or months. Many edentulous people kept their food in the mouth for a long time and sucked it, and so did much more to insalivate it than did other people with teeth. He had no details to give with regard to crowns. When the mouth was septic, the whole 25 ft. of intestine was kept septic as well. Dr. Midelton had taken up a particular form of counter-irritation, but he (Dr. Ackerley) did not think the pyogenic organisms had anything to do with the good results which he obtained; as far as he was using such organisms, he was doing harm, not good. He had been connected with an isolation hospital, and when the patients with diphtheria had clean mouths and good teeth, the pathologist's report on swabs from the throat was that there were pure, or almost pure, cultures of the diphtheria bacillus, but when the mouth was dirty there was a mixed infection and the type of disease was severe. In cases of typhoid, too, it made an enormous difference whether the mouth was clean or dirty on the course of the disease. But there was a danger of attributing too much to oral sepsis. A good

112 Ackerley: *Condition of the Mouth in Chronic Disease*

paper had appeared in the *Lancet* last year, by Dr. Wirgman and Mr. Watson Turner, on "Local Sepsis as a Factor in Rheumatism and Gout."¹ It was a factor, but it should not be elevated into too much importance and regarded as the sole factor in these diseases. The physician should seek to remove *every* factor of disease, and he should not treat the local sepsis and nothing else. He suggested to Dr. Midelton that he should treat the oral sepsis concurrently with the counter-irritation for the rheumatoid condition. On one point he would have liked the opinion of the President of the Section—namely, as to whether dentists considered that it was wise to keep plates in the mouth at night. He asked, because many patients assured him that the dentist told them to keep the plates in through the night.

The PRESIDENT replied that the practice of keeping the plates in at night was altogether bad.

¹ *Lancet*, 1909, ii, p. 1665.

Odontological Section.

April 25, 1910.

Mr. W. HERN, President of the Section, in the Chair.

Two Cases of Suppuration in the Maxilla.

By CARL SCHELLING, L.D.S.

ON May 17, 1909, I was sent for by Dr. Ingram to see a lady whose face was too much swollen for him to allow her to leave her room. Two weeks previously she had been to a dentist who had removed the left upper bicuspid and third molar, but informed her that a small piece of one had been left behind; she did not, however, know of which. I suggested that the gentleman who had previously seen the case should be again consulted, but was informed that he had declined to attend, being too busy. On examination of the mouth I saw the very nearly healed socket of the left upper molar, and could find no fragment of root with a fine probe. A gap anterior to the first molar was also found, but the gum was hard and of good colour there. The swelling extended so far along the left sulcus that it could not be said to be over any one tooth more than another, and all appeared sound. On feeling high up a slight elasticity was felt over the bicuspid region, and, after refrigerating the part with ethyl chloride, an incision was made and a great quantity of thick pus was evacuated. After some days, under the care of Dr. Ingram, the patient was sent to Mr. Clarke, who made the radiograph I now show, and it was then an easy matter to incise the gum, which I did under gas on June 10, and removed the small piece of root which had caused the trouble.

In the second case, a lady, who gave the history of having had a very swollen face, and a difficult extraction performed some months ago in India, stated that a fortnight previous to her appointment she had been kept at home for several days with a return of the same trouble.

114 Schelling: *Neuralgia due to Impacted Wisdom Teeth*

Posteriorly to the left upper canine only a molar remained, and a small fistulous opening some way anterior to the molar led up to some dental tissue. Pus had been discharged from the nose, so that the antrum was possibly affected. I sent the patient to Mr. Clarke, who made the radiograph, and informed me that although the fragment at the end of the sinus was in relation with the antrum, yet he was more disposed to suspect a curved anterior root of the pulpless upper molar as being the cause. Under gas the small fragment was removed, the forceps, however, breaking into the antrum when being pressed up; but this was not unexpected by me. The molar was then carefully taken hold of, but came out, leaving the tip of the curved root behind. A small scaler was passed up through the first molar socket into the antrum and the portion of root was found and hooked down. A vulcanite plug was adjusted to the denture plate worn by the patient, and after some days' treatment by irrigation with antiseptic douches it was cut off and the opening allowed to heal. Some weeks afterwards the patient returned somewhat alarmed, as she had been able to force some air through the opening. This was touched with lin. iodi on cotton wool wrapped round a bristle, and I now hear that the patient has had no more trouble and has returned to India.

A Case of Neuralgia due to Impacted Lower Wisdom Teeth, in which Skiagrams helped the Diagnosis.

By CARL SCHELLING, L.D.S.

AT the conclusion of a series of visits, in which I believed myself to have thoroughly put a young lady's teeth into good order, I was somewhat surprised by her mother inquiring whether my work would have any effect on the severe neuralgia and occasional swelling of the neck from which the daughter suffered. As I had only filled simple cavities, and had no notion of any exposed nerves or pulpless teeth, I asked her to resume her place in my chair, and again made a complete examination. The pain was said to be worse on the left side and to come on without warning at any time, so that often the patient had to leave the dinner table or a dance. As I found nothing beyond the absence of the wisdom teeth, I suggested that Mr. C. A. Clarke should be asked to take skiagrams, and when this was done they showed the impacted lower third molars very well. After I had removed the

second molars the neuralgia disappeared. I have lately seen the lady, and she is still free from pain. For the last three years she has been under the treatment of an aurist, to whom I sent the skiagrams and a note of the treatment adopted, and he expressed his approval to me.

Two Cases of Suppurating Dental Cysts invading the Maxillary Antrum.

By HERBERT TILLEY, F.R.C.S.

Case I.—Mrs. B. W., aged 27, consulted me on January 26, 1910, complaining of "neuralgia of the left cheek" which had lasted for six weeks. She said that a diseased tooth had been "filled," but that the filling had been removed without any relief from the neuralgia, and that during the period stated she had occasionally noticed an unpleasant nasal discharge from the left nostril. Transillumination showed that the left antrum was dark, but on exploration of the sinus through the nose by means of a trocar and cannula passed through the inferior meatus it was found that no fluid could be made to return from the nose. It was therefore obvious that the cannula was not in the antral cavity proper, or, if it were, that the natural opening of the antrum into the nose was closed—a rare condition. On January 28, assisted by Mr. Betts, I removed the anterior two-thirds of the left inferior turbinal and opened the antrum by a large opening in the canine fossa, when a quantity of foul pus escaped. The cavity opened was the size of a normal adult antrum; it was lined with red, vascular granulations, and proved to be a large bony-walled cyst which had completely filled and moulded itself to the antral cavity. The cyst wall was removed piecemeal through the opening in the canine fossa, and a large opening was then made into the nose by the removal of the greater part of the inner antral wall. The bucco-antral wound was sutured by two interrupted horse-hair stitches, and the patient made an uninterrupted and rapid recovery.

Case II.—Major K., aged 35, was sent to me on February 2, 1910, because of a "puffy swelling under the lower part of the right cheek" from which a discharge of pus into the mouth had taken place for at least two months. The second upper right bicuspid had been removed, but the swelling over the alveolus remained and varied in size according to the freedom with which pus discharged from the alveolar fistula.

Aching pain in the right cheek was more or less constant. Transillumination showed that the right antrum was dark, but here again, as in Case I, it was impossible to obtain a return of fluid when intranasal irrigation of the antrum was practised. On February 9 the same operative procedure was carried out as in Case I, and with an equally successful result.

The cases are interesting in that they illustrate how large these cysts may become, and they may afford an explanation of the persistence of some alveolar fistulæ even when the diseased tooth has been removed.

From the nasal surgeon's point of view the prognosis in each patient was interesting, because it was quite obvious that when the antrum was perforated and no fluid could be injected, one must be in a closed cavity such as a cyst, or, if in the true antrum, that its natural "ostium" was not patent—a rare condition. For such reasons a suppurating cyst was diagnosed in each case. Equally important is the question of treatment. To open the cysts from the alveolar aspect, curette their lining membrane, and to pack with gauze until the cavity is obliterated by granulation tissue is scarcely fair to the patient, and is certainly meddlesome interference on the part of the surgeon; and yet there are still text-books which advise this as the correct treatment.

One cannot conceive that either of these bony-walled cysts would have ever become obliterated, but it is easy to imagine what a constant and wearisome nuisance it would have been to the patients who, once or twice daily for month after month, would be compelled to pack these suppurating cavities every day, and whose meals would possibly have been flavoured by "sauce iodoformée."

By removing the cyst wall entirely, making a large counter-opening into the nose, and suturing the bucco-antral mucous membrane, the patient is practically well in a week, and for purposes of cleanliness need only irrigate the nose twice daily for the following two or three weeks.

Mr. HERBERT TILLEY exhibited a series of diagrams by which he explained the method of operation, and added that on the preceding Thursday morning a patient came to his clinic with a dental cyst which had been packed for six months; one could still pass a probe $1\frac{1}{2}$ in. into it. He thought it would just illustrate the inefficiency of that method of treatment if he brought the patient to the meeting for members to see.

The condition of the lady was exhibited to the members.

As far as one could see, the cyst would be there for another six months or even six years. It was really a simple treatment to give the patient a general anæsthetic, remove the anterior and inner antral walls, and thus induce a rapid and complete recovery.

DISCUSSION.

In reply to the President, Mr. TILLEY said that his experience of the success of the operation in the treatment of chronic suppuration had led him to employ the same method in dealing with large suppurating dental cysts. The diagrams which he showed were made to illustrate the radical treatment of chronic antral empyema by freely draining the nose, but the same treatment was efficient for a *large* cyst which also filled the antrum. In reply to Mr. J. F. Colyer, he said that one of the patients had noticed an unpleasant smell, but he thought the pus had escaped from the large cyst into the antrum and had been discharged into the nose. In the other case there was no communication with the antrum at all; it was merely a very large cyst filling that sinus. It occurred to him to remove the whole bony wall and leave the antrum intact, making a counter-opening into the nose for the sake of securing free and permanent drainage. In reply to Mr. James, he said that he was not conversant with the different types of dental cysts, and could only say that the two he had recorded appeared to him to be alike. In the first case he thought when he gained access to the cyst that he was in the antrum, but while mopping it out he saw the whole surface move. The cyst was as large as a good-sized walnut and consisted of a very thin bony wall. Those who had studied this subject in the museum of the Royal College of Surgeons would have seen there a beautiful dental cyst almost completely filling the antrum, and the cysts he had referred to seemed identical with that. On the outer surface of the cyst at one point was a polypus which seemed identical with those met with in the nose.

Mr. SCHELLING said, in regard to the first case mentioned by Mr. Tilley, he believed the patient was a patient of his partner, Mr. Betts. Mr. Betts had filled a tooth, but the patient came back with the pain not at all relieved. He believed Mr. Betts killed the pulp in the molar. He looked the matter up, and saw that it had been done thoroughly according to the book. He took out an old filling from the next tooth and cleared out the palatine root. Being short of time he could not do anything with the buccal roots, and just put a dressing in and told the patient to come and see Mr. Betts in a few days. On that occasion he believed Mr. Betts opened up in the direction of the anterior buccal root and came upon the canal, from which a large quantity of fluid issued. He believed Mr. Betts said that he soaked something like twenty or thirty large plugs of cotton wool with the fluid from the cavity, but whether that collection of pus was in the antrum or not he did not know.

Mr. J. F. COLYER wished to put in a word for the treatment from the mouth of dental cysts invading the antrum. At the Royal Dental Hospital a very fair number of such cysts were seen, and where the cyst invaded the antrum most excellent results could be obtained by opening the cyst wall, removing it very freely, and making a surface draining into the mouth—just as

easily, in fact, as a surface drain could be made into the nose. The whole secret consisted of entirely removing the cyst wall and draining into the mouth so as to leave a self-cleansing surface.

The PRESIDENT (Mr. Hern) thought there was a distinct advantage in draining an antrum or a suppurating cyst into the nose instead of into the mouth, because the more free the mouth could be kept from pus the better.

A Case of Painful Attrition.

By WILLIAM RUSHTON, L.D.S.

ATTRITION, or the wearing down of the masticating surfaces of teeth, is a condition which usually gives rise to so little discomfort that most of the text-books ignore it, or only mention it casually. The cases we usually see are those in which, the molars having been lost, mastication is performed by the anterior teeth, which consequently become much worn down. We also occasionally find molars the enamel of which has partially or totally disappeared, and the dentine, where exposed, gives more or less discomfort on mastication. There are also cases—chiefly in the temporary dentition—where active caries has by some means become arrested, and where the dentine becomes black, polished and worn down to the gum level, without apparently causing any discomfort to the patient. (Models of child's teeth passed round.) Where the condition gives pain it is usually treated by the application of silver nitrate, by filling, crowning, or by supplying an artificial denture to relieve the wear of the natural teeth. I once saw an elderly American gentleman who had had every tooth in his head encased in gold. The treatment was effectual, but it imparted a Midas-like expression to his smile which was far from pleasing.

The case I bring before you to-night is remarkable for the large amount of molar attrition, especially in the mandible, for the comparative youth of the individual, and for the great amount of suffering caused. The subject is a dentist, an old fellow-student of my own, aged 41. He is an Armenian, practising in Syria. I pass round the models of his teeth, which he has sent me, which show very clearly the manner in which his molars are worn down. He complains of great pain, especially in the lower molars on mastication. They are also very sensitive to sweets, acids, and to thermal changes. The teeth are

otherwise perfectly sound, with the exception of the right lower second bicuspid, which has an amalgam stopping and seems to give no pain. He has frequently applied silver nitrate with no good result. Twenty-two years ago I examined his mouth and there found that his first lower molars were somewhat worn down, but not painful. The second molars were unaffected at that time, and have given no trouble until comparatively recently. But during the last two or three years attrition has proceeded with great rapidity in the second molars, but seems to be making little or no headway in the first molars. After making inquiries as to his food and habits, he replied that cases of attrition were fairly common in Syria "amongst Turks and Christians, poor and rich, high class and low class," but he has not met with anyone who suffers in the manner he does. He does not admit that food is the cause, but admits that he gnashes his teeth during sleep, and cannot break himself of the habit. This, I think, must be the cause of his trouble, possibly aided by the composition of his teeth or the state of his oral secretions.

It would be interesting to inquire why the second molars have gained so much on the first in the wearing-down process, and why the mandibular molars are more abraded than the maxillary ones. One would have thought they would all have been equally affected.

The treatment under ordinary circumstances, I take it, would be either to devitalize and extirpate the pulps of the sensitive teeth under an anæsthetic, or to protect them by crowns. The difficulty in his case is that he is the only dentist in that part of Syria. He admits that to grind his teeth for crowns fills him with terror, and possibly he would find exposing his pulps equally objectionable, if not more so. I have advised him to construct for himself a biting-plate, to be used at meals and during sleep, adapted as closely as possible to the lower molars and to the articulation. I have not yet heard whether he has tried this or found it successful, but if any member can suggest any other treatment to relieve an isolated and suffering *confrère*, I shall be delighted to communicate the same to him.

Before concluding this short communication I should like to say a few words about a remarkable collection of Egyptian skulls which were exhibited at the Royal College of Surgeons' Museum last year. These skulls were of an early date, and evidently belonged to the labouring class. The teeth were described in the College catalogue as being extensively carious, and the moral was drawn that caries was as common amongst the ancient Egyptian poor as amongst our poor of to-day. I carefully examined those skulls, and could find no dental caries, though

I saw evidence of alveolar abscesses. What was supposed by the compiler of the catalogue to be caries was extraordinary attrition, and it is quite possible that this was also the cause of the abscesses, consequent on exposed pulps. To that extent at least we must be grateful for the less gritty food of to-day.

DISCUSSION.

The PRESIDENT asked whether the patient, as a child, was addicted to grinding its teeth at night.

Mr. F. J. BENNETT did not quite understand how food could have made the lower teeth wear at a greater rate than the upper ones.

Mr. J. F. COLYER said the question of grinding the teeth was an interesting one, and, in the opinion of some, was frequently seen in individuals suffering from cerebral disease. The name "bruxomania" had been given to the condition. There was very little doubt that children affected with any kind of cerebral irritation had a tendency to grind their teeth. Recently he had had a case which troubled him of a young lady who had been grinding her teeth down very severely. He tried a biting block, but it only made matters a good deal worse. He came to the conclusion that a good deal of her bruxomania was due to a certain amount of periodontal disease, and directly he treated her for general periodontitis she to a great extent got rid of the grinding of the teeth. It would not be an unwise thing for Mr. Rushton to suggest to his friend that he should get rid of the possibility of any periodontal disease.

Mr. GABELL said he had met with a case of a child who had worn all his teeth down to the gum; there was no mental abnormality, and the second dentition had been absolutely normal. He had also had a case of attrition, similar to Mr. Rushton's case, in an extremely muscular man. So bad was the grinding of the teeth that his wife could not sleep for the noise. The case was treated with a velum pad, which was fitted over the teeth, and adapted to the bite of the upper teeth, keeping clear of the gums of the lower, and the plate was worn at night. Small holes were bored in the plate, which was tied to the button of the pyjamas, so that the patient could not swallow the plate in the night. The teeth were normal, and the mouth very clean and healthy, and there were a large number of gold fillings. Of recent years the trouble had ceased and the plate had been dispensed with.

Mr. STANLEY MUMMERY mentioned the case of an Oxford professor who ate a great many apples, five or six a day, and had the condition referred to by Mr. Rushton very strongly marked. Some of the teeth were cut right down half way through the pulp. The patient did not suffer much from sensitiveness, except in one or two of the teeth, and these were crowned with gold caps. A few years ago he married, and his wife developed a similar tendency, and her

teeth were now getting nearly as bad as her husband's were; he had learned from her that she had also taken to excessive apple-eating. He could not help thinking that the acid apples had a great deal to do with the condition.

The PRESIDENT said he had frequently noticed that when attrition affected the lower molars, a cupping of the masticating surface of these teeth took place in the positions of the cusps, whereas the central portions of the crowns were affected to a much smaller extent. The condition was well shown in the model of Mr. Rushton's case.

Mr. RUSHTON thought there was probably a good deal in what Mr. Colyer had said. With respect to the child's models, the patient had not been a normal child, and had been very rickety, but he was not cerebrally affected. With regard to attrition being a manifestation of periodontal disease, it might sometimes be so; but he was told by his friend that his mouth was perfectly healthy in every way. With regard to apple-eating, it was quite likely that the acid might have some effect, and he had seen a case in which a lady sucked a large quantity of lemons and had a somewhat similar condition. But why should the lower teeth be affected more than the upper, and the second molars more than the first?

Two Odontoceles, and some other Cysts.

By A. HOPEWELL-SMITH, M.R.C.S., L.D.S.

INTRODUCTORY.

THE two cases which are about to be narrated, possessing a few common characteristics or relationships, are rarely seen, and are very dissimilar from a pathogenetic point of view. They are worthy of record as being extremely remarkable and interesting. They are also of sufficient importance to illustrate the belief that greater precision is required of dental surgeons when describing certain pathological changes that may take place in the osseous framework of the maxillæ and mandible.

This communication has been written around these two cases, and seeks to establish the opinion that there is room for an amplification and revision of the nomenclature employed in the subject of dental pathology, and that new terms must necessarily be introduced to more accurately represent conditions as our knowledge of the special pathology of the teeth advances. It further endeavours to throw some light on the obscurity of the origin of cysts of the jaws, and to ascertain in what circumstances or through what agencies fluid is produced in such

amount as to constitute the chief clinical characteristic. The signs and symptoms are unimportant and uninformative, but the morbid conditions and the patho-histology of the tissues are of extraordinary significance. If the deductions arrived at are inconclusive, or open to different construction from that which I am able to supply, allowance must be made for the difficulties attaching to the research and to the great mystery surrounding the unique display of morbid phenomena.

It will be found that, although placed in juxtaposition under the heading which gives the title to this paper, it is possible that, if the abnormal changes have been thoroughly comprehended and adequately interpreted, each case can occupy a sub-division of such a title. Thus the first would be designated an example of a sub-capsular odontocoele, and the other of an extra-capsular odontocoele. They merely fall into the same main category because, in the opinion of the writer, they cannot strictly and scientifically be placed under any other classification. A common feature here is the existence of a unilocular cyst in the jaws, containing an anomalous tooth—a canine, non-erupted, and so-called “encysted,” disclosing itself in adult life. It is incorrect to describe “a case of an encysted tooth”; the fact that it is imbedded in the bone does not of necessity mean a disease of the tooth or jaw, but a state of being or condition in which the tooth happens to exist. Similarly, it is incorrect to speak of dental caries as a disease. *Pyorrhæa alveolaris* is not a malady, though commonly described as such, but a symptom, a manifestation of a disease or diseases; and an encysted tooth is not necessarily an affected organ.

We are led to understand, from the pages of text-books and current literature, that a buried, “imprisoned” tooth, as it is somewhat fancifully called by Roswell Park [8] in “The Principles and Practice of Modern Surgery,” 1908, may induce a passive or active reaction to the containing tissues. Thus (1) it may, usually, remain in its uncommon irregular position undisturbed, unnoticed, inert, impacted or otherwise throughout the life cycle of the individual, giving rise to no signs or symptoms of anomaly; or (2), it may, rarely, originate a tumour, through the production and accumulation of a fluid body around it. Clinically, when a tense, fluctuating, non-inflammatory, painless swelling of the jaw presents itself for diagnosis, either a dental cyst or a follicular odontome is brought to mind. These two, and especially the former, are the commonest cysts with which the dental surgeon has to deal. But other species of cysts may occur at times, and it is possible to fully enumerate them in their probable order of frequency.

SPECIES OF CYSTS OF THE JAWS, BASED ON THEIR PATHOLOGICAL VARIATIONS.

- (1) Dental cysts.
- (2) Eruption cysts.
- (3) Follicular odontomes.
- (4) Epithelial odontomes or multilocular cystic tumours.
- (5) Mucous cysts of the antrum.
- (6) Odontoceles: (a) sub-capsular, (b) extra-capsular.
- (7) Cystic adenomata of the antrum and gum.

Of these, I propose to discuss briefly the first three, fully the odontoceles, sparingly the cystic adenomata, and not at all the antral mucous cysts and epithelial odontomes, and, as time is short, to view them chiefly from the standpoint of their ætiology and pathology.

Dental Cysts.

With regard to the evolution of a dental cyst, we have learnt to consider that it is of inflammatory origin, being due to proliferation, death, and subsequent liquefaction of the cells which form the *débris épithéliaux paradentaires* of Malassez—the vestigial remains of the epithelial sheath of Hertwig—in the periodontal membrane, the consequence of an infection by pyogenic micro-organisms of that tissue. This is therefore an example of an acquired lesion.

A dental cyst, as dental surgeons understand the term, does not conform at all closely to the lines of classification given by certain general pathologists and surgeons. In the first stage of its growth, it is not derived from a distension of pre-existing cavities or spaces such as are a bursa, a ganglion, a ranula, a galactocoele; it is not a cyst of new formation, like an adventitious bursa, a hæmatoma, a proliferous compound cyst, a parasitic (hydatid) or an implantation cyst, neither is it of congenital derivation, as is, for instance, a dermoid cyst or a cystic lymphangioma. The important point to emphasize is that it owes its being to an inflammatory condition of the tissue in which it is found, whereas none of the cysts alluded to above directly does.

By custom we associate the term with this pathological condition; but probably if a greater degree of refinement and exactness of expression were insisted upon we should conclude that it is, therefore, a form of periosteal (periodontal) cystic degeneration, or a periosteal

maxillary cyst, as suggested by Partsch. It will be immediately patent to the impartial mind, on hearing the clinical histories of the morbid anatomy of the special cases, that neither constitutes the so-called dental cyst.

Follicular Odontomes.

Follicular odontomes, on the other hand, are undoubtedly congenital or developmental in their origin. But a good deal of confusion still exists as to their true pathogenesis. It is unnecessary to enter into the arguments of many writers. Let it suffice therefore to recall that, among the older ones, Broca [3] believed that they arose within the tooth capsule, sac, or follicle, the enamel organ having disappeared "under morbid influences"; that Malassez [6] explained that they were due to hypertrophy of epithelial rudiments of the enamel organ; that Albarran [1] considered they were inaugurated by the proliferation of the aborted epithelium of the fibrous tissues normally present in the *inter dentis*; and that Salter [9] ascribed to "a sort of epithelium" clothing the so-called enamel pulp "the power of assuming the function of secreting fluid."

Bland-Sutton [2] defines them thus: "Swellings [which are] often called dentigerous cysts, a term which has come to be used so very loosely that it should be discarded in the necessity for precision. They arise commonly in connexion with teeth of the permanent set, and especially with the molars; sometimes they attain large dimensions and produce great deformity, especially when they arise in the upper jaws and happen to be bilateral. Rarely they occur in connexion with supernumerary teeth. The wall represents an expanded tooth-follicle . . . The cavity of the cyst usually contains viscid fluid and the crown or the root of an imperfectly developed tooth. Occasionally the tooth is loose in the follicle, sometimes inverted, and often its root is truncated; exceptionally the tooth is absent or represented by an ill-shaped denticle. The walls of the cyst always contain calcific or osseous matter; the amount varies considerably."

Tomes and Nowell [10] write: "The follicular cyst arises in connexion with teeth retained in the jaws, generally bicuspid or molars. They cause great distension of the jaws" (p. 725); and, in accounting for their mode of formation and generation of the fluid contents, assert (p. 732): "When the development of the enamel is completed, its outer surface becomes perfectly detached from the investing soft tissue, and a small amount of transparent fluid not uncommonly collects in the interval so formed."

It is a well-known fact that the deciduous and permanent teeth, when about to erupt, may present over their crowns a bluish, soft vesicle containing serum (eruption cysts). It is not an easy matter to explain the presence of the vesicles over the *teeth of succession* because of the presence of the absorbent organ, and nearly always follicular odontomes are formed in connexion with the members of the permanent series. The authors just cited believe that this fact—viz. the occurrence of vesicles—furnishes an explanation of the manner in which cystic tumours containing unerupted teeth arise. "Fluid collects between the enamel and the tooth capsule," they say. Assuming this statement to be correct, and although no mention is made of the enamel cuticle, we are led to ask "Whence comes the fluid? Why does it collect?" And we are told that fluid is normally and universally present over the crowns of unerupted teeth. If this were so, one would expect to find that follicular odontomes were extremely common, which they are not, and that unerupted teeth could never remain in an innocuous state in the bones of the jaws, which they do.

Paul [7], writing in 1894, observed: "On dissecting them (i.e., the teeth of sheep and monkey in their sacs), it appears that at a certain stage the crown of the tooth was quite free inside the sac, but that at a slightly later stage the sac adhered to the tooth, although it could be easily stripped from it."

These authors proceed: "As the cyst enlarges, the contiguous bone is removed to make room for it, fresh bone being concurrently deposited on the outside of the jaw. In the case of such a cyst lying in front of a tooth which is being cut, it is obliterated by the advancing tooth or it bursts; but when situated deeply in the jaw, a cystic tumour may be the result."

Finally, Heath [4] defines follicular odontomes as "cysts [which] contain one or more teeth in their interior or in their wall. The teeth may be well formed or may be quite rudimentary, consisting of irregular masses of bone and enamel."

The Origin of the Cystic Fluid.

Elsewhere I have expressed the opinion that the fluid contents of these tooth-bearing cysts is derived from the degenerated cells of the stellate reticulum of the enamel organ. Instead of becoming absorbed in the usual way, they may, for some recondite reason, not disappear, but may further degenerate and liquefy, and produce a potential cavity

which, on being filled with fluid, is the beginning of the formation of the cyst. The idea is apparently acceptable to Bland-Sutton, or at all events thought by him suitable for reference in the last edition of his work on "Tumours, Innocent and Malignant."

Reverting for a moment to the case which prompted the publication of this view, the patient was a boy, aged $9\frac{1}{2}$, who had a follicular odontome in connexion with the first mandibular left premolar. Immediately after the tooth had been removed and the necessary surgical toilette completed, the premolar was subjected to a chemical and microscopical test to ascertain whether Nasmyth's membrane was present, and, if so, whether it was hyperplastic and diseased—an independent investigation undertaken solely for the purpose of discovering whence came the cystic fluid. Phloroglucin and nitric-acid mixture, freshly made, was employed. Neither the translucent pellicle nor the cellular layer could be raised from the surface of the partially-decalcified enamel. They were absent. After the publication of a short note to this effect, it transpired that Mr. Maggs and Dr. Pare had also failed to find Nasmyth's membrane in a similar case, my attention having been drawn to the fact that a paper by them had already appeared in the *Guy's Hospital Gazette* for 1894. They should therefore be credited with the original observation. The result of my research was further gratifyingly, though unexpectedly, corroborated by the material supplied by a case operated upon by Jordan Lloyd, of Birmingham, and communicated privately to me, in which—for the third time—it had been noticed that the enamel cuticle was completely absent from the teeth in cysts formed in such a manner.

The pathological controversialist might, however, possibly argue that liquefaction of these cells does not occur, and that, even if it did, it would not account for the loss of the inner layer. To that my answer would be twofold: First, if the liquefaction (that is, the production of a fluid) took place at the time just antecedent to the date when the ameloblasts were about to finally undergo conversion into the pellicle, it is reasonable to suppose that the mere presence or pressure of the fluid might inhibit this function and destroy the cells. Again, supposing that it be pointed out that the atrophy of the stellate reticulum cells could not induce a liquefaction, if a small potential cavity were formed during their disappearance, there might, as a consequence of the negative pressure, be a slow effusion of serum or lymph into the space so created from the numberless blood-vessels lying in the immediate neighbourhood of the enamel organ.

Again, it might be pointed out by the critic that the cystic fluid is commonly too great in amount to be able to originate in the breaking down of a few stellate cells, and that its source must be sought for elsewhere. To this it may reasonably be argued that a combination of the two factors—viz., the production of a space and effusion into it of lymph from the vessels—would account for it. If this were not so, one would have to bring forward evidence of the existence of a secretory epithelial lining to the cyst wall; and this I have not been able to do.

Histological Notes.

As bearing on the pathology of these cases, it is necessary to recall as succinctly as possible the anatomical topography of the parts, and to explain the grounds for my belief that the liquefaction of the stellate reticulum would account for the fluid in a dentigerous cyst. The enamel organ at maturity consists of four different cell elements, arranged from within outwards, as the (1) internal epithelium or the ameloblasts—elongated, columnar cells, measuring $15\ \mu$ to $20\ \mu$, set in immediate apposition; (2) the *stratum intermedium*, a narrow layer of small polygonal cells; (3) the stellate reticulum, mucoid cells with round nuclei and numerous long branching processes; and (4) the external epithelium, a layer of single rounded or flattened cells. The functions of these are supposed to be as follow: The first to form enamel, and, when "spent," the translucent pellicle, or inner layer of Nasmyth's membrane; the second to recruit or rehabilitate the ameloblasts; the third to act as a "packing" material to the enamel organ; and the last (somewhat doubtful, but believed by some—Professor Paul, for instance) eventually to constitute, on persistence, the cellular layer of the enamel cuticle. Now, outside this external epithelium, which, it is important to note, is in direct continuity with the fibrous tissues which form the dental capsule—there is no sharp line of demarcation between the two, though in speaking of the two structures one unconsciously dissociates them—comes the follicle or tooth-sac itself. Composed, *when young*, of fibrous tissue with a feeble supply of round cells, it contains numerous gland-like epithelial bodies, which I first observed in 1900, to which allusion was made last year by Mr. Warwick James in a paper on "Eruption" read before this Section.

It is therefore clear that the dental capsule usually has, on its internal aspect, a layer of epithelium—viz., the external epithelium of the enamel organ (fig. 1). It is possible, in fortunate circumstances, to show the

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It is therefore clear that the dental capsule usually has, on its internal aspect, a layer of epithelium—viz., the external epithelium of the enamel organ (fig. 1). It is possible, in fortunate circumstances, to show the

lining of the capsule. To give a concrete example: If an unerupted first premolar be removed at the age of 7 to $7\frac{1}{2}$, and its soft-tissued investment allowed to remain *in statu quo ante*, on making vertical sections one can see, under the $\frac{1}{8}$ -in. objective, the ameloblasts becoming converted into the translucent pellicle of Nasmyth's membrane. At the cervical margin of such a tooth they are elongated and cylindrical, but little altered from those of activity—merely shorter. Higher up, nearer the cusps of the crown, however, they are shrunken and flattened, and while still retaining their prominent nuclei, often become hexagonal or pentagonal in outline. In the neighbourhood of the extremities of the cusps they have become fused to form a homogeneous membrane—the pellicular or inner layer of Nasmyth's membrane. Next to them is a single flat layer of cells, the external epithelium closely applied to their surface. Though so intimately approximated there is a wide range of difference from an embryological point of view between the two, the external epithelial cells being epiblastic and the dental capsule mesoblastic in origin.

Now, if the stellate cells of the enamel organ undergo further softening, disintegration and liquefaction, the first stage of the formation of a follicular odontome is inaugurated. Little by little the fluid collects, until a cyst is produced, with the external epithelial cells on the outside and the internal epithelial cells on the inside. The first form a definite layer of compound epithelium and are not secretory cells. The second has sometimes not had an opportunity of becoming metamorphosed into the pellicle of the enamel cuticle (this is quite conceivable, and, I hold, a common-sense view) on account of the accumulation of the fluid; hence its absence on three authenticated occasions. At other times the fluid has probably collected *after* the production of the pellicle had occurred, but the cellular layer, instead of appearing on the surface as part of Nasmyth's membrane, remained as the adventitious lining of the cyst wall.

Another fact in agreement with the opinion which is being postulated is the striking anatomical resemblance between the degenerate cells of the stellate reticulum and the degenerating cells of an epithelial odontome¹ (fig. 2).

¹ The phenomena associated with this degeneration were thoroughly described by Eve in an important paper on "Cystic and Encysted solid Tumours of the Jaws, with Observations on the Structure of the Enamel Organ," which he read before the Odontological Society of Great Britain in 1885. From his remarks it was apparent that he had corroborated the results of the researches of Falkson and Bryck of Germany, who had independently arrived at similar conclusions.

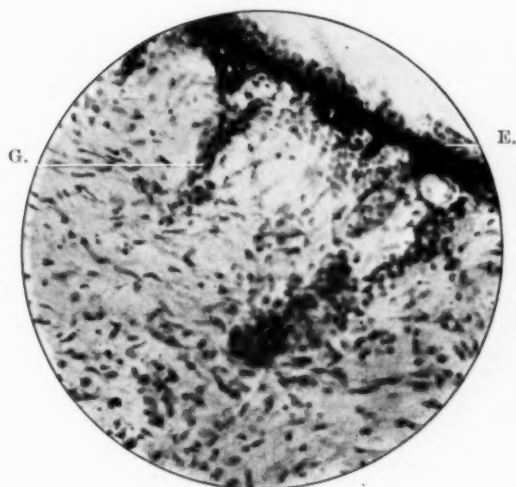


FIG. 1.

Vertical section of dental capsule, stained with haematoxylin only. G., gland-like bodies; E., epithelial cells lining internal surface ($\times 240$).



FIG. 2.

Epithelial cells in an epithelial odontome, becoming stellate in appearance and breaking down into a colloid mass ($\times 240$).

The Dental Capsule.

It is, next, important to examine the histological elements existing in a mature dental capsule, and outline its brief life-history. The word "follicle" is employed at times in this paper, although it does not very scientifically express its nature and functions; it is neither the homologue nor the analogue of a hair follicle, a sebaceous follicle, a mucous crypt of the tonsils, or an agminated follicle of the small intestines. I have been unable to find another organ in the body which is its counterpart. It exists for a few years only, and when its work—that of protecting the crown of the erupting tooth, after being instrumental in generating the cementum and periodontal membrane—is accomplished, it disappears entirely, completely differing from such organs as the uterine adnexa, which persist after the cessation of their functions, and even the thymus gland, which, as a rule, leaves traces behind. It can be fairly claimed for the dental follicle that in this respect it is unique. Regarded from an embryological aspect, the dental capsule, say, of the first premolar, may be observed in certain portions of the oral submucous and alveolar tissues about the ninetyeth day of intra-uterine life in man, and can be well demonstrated in sagittal sections of the jaws of kittens three weeks old. The cells are very elongated and thin, with small lenticular nuclei, chiefly arranged in longitudinal bundles corresponding to the long axis of the tooth germ. Later on they become developed into the extended fusiform cells of fibrous connective tissue. Eventually the capsule undergoes atrophy and degeneration by the loss of the nuclei of its cells and vacuolation of its substance, in this specific instance, about the seventh to the ninth year in man (fig. 3). This vacuolation, it is important to notice, occurs as a normal change in the sacs of teeth about to erupt, of course just prior to their disappearance; but when a tooth is retained *in situ* in the bone, it does not follow that it undergoes this vacuolation. In the two special cases it has not done so, although the patients were aged 31 and 41 years respectively.

In addition to the connective-tissue fibres of maturity, numbers of small, discrete masses of epithelium are seen. It is very probable that many of these beget the mucous glands which lie scattered about the subepithelial tissues of the gum.

[Let me here stop to call your attention to a new fact which I have ascertained only within the last few weeks. There are large mucous glands in the gum at the cervical margins of the teeth on the *lingual* or *palatal* side (fig. 4), none in that on the *labio-buccal* aspect of the alveolar



FIG. 3.
Dental capsule vacuolated ($\times 60$).

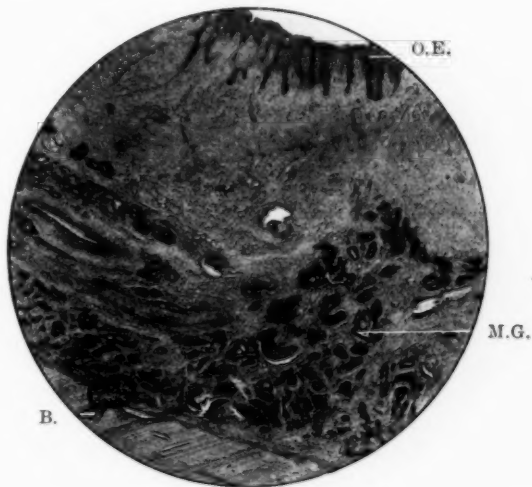


FIG. 4.
Sagittal section through tissues lying over the external aspect of the alveolar process. B., bone of jaw; O.E., oral epithelium; M.G., small portion of a large mucous gland ($\times 45$).

process. I experimentally proved this by excising a portion of normal gum immediately in contact with the teeth, on the outer side of the alveolar process of the mandible of a man, aged about 44, and chose a spot between the two right premolars. It measured 6.5 mm. in length. There was no mucous gland to be seen. Similarly none was present in the gum on the *labial* aspect of the bone over the root of the second left maxillary incisor of a woman aged 25. Again, in a piece of the papilla, over the septum which intervenes between two mandibular molars, I found no mucous gland. The reason of this, I think, is obvious. Mucous glands are not wanted in the former situation, because of the existence of the countless numbers which open on the free surface of the lips and cheeks, and in the latter situation because of lack of room.]

The majority of the epithelial bodies, derived from the fenestration of the Zahnleiste, may give rise to the eruption cysts or epithelial odontomes, supernumerary teeth, &c.

It is also conceivable that for some reason or other the cells of one or more of these gland-like bodies might undergo karyokinesis, and, like the paradental "rests," though in consequence of a dissimilar stimulation, might rapidly multiply, form large epithelial masses, of which the central cells, cut off from their nourishment, would die, degenerate, and liquefy. Here, then, a cyst might form—a capsular cyst—but if it were evolved from the central portions of the dental follicle, there would ultimately be found two layers of heteromorphic cells in the perfected follicular odontome wall; and this is never so. A follicular odontome is therefore an example of a congenital anomaly.

It will shortly be satisfactorily shown that neither of the cases to be now detailed furnishes histological or pathological evidences of being a follicular odontome *per se*.

A Sub-capsular Odontoccele. (A) The case of Mr. R. L.

This patient, aged 31, was sent to me in January, 1909, by his dental surgeon, whom he had consulted about a circumscribed progressive enlargement of the jaw. On examination a large, smooth, bluish, fluctuating, painless distension of the bone was seen occupying the right canine region of the mandible. Its presence had been detected subjectively only five weeks previously. The canine was missing, and there was no history of its having been extracted, though several teeth had been removed and a denture was being worn. The first premolar was almost in contact with the distal surface of the lateral incisor. The

corresponding tooth was not in evidence, but both left canines were erupted in correct alignment with the dental arches. A cyst was diagnosed. Radiographs disclosed the following condition of the parts: At the base of an extensive cavity in the bone, measuring 2.5 cm. by 2 cm. by 1.5 cm., was a canine, non-erupted, non-impacted, non-absorbed. It was placed vertically in the jaw, its crown pointing upwards and extending into a somewhat triangular hollow, produced by the divergency of the roots of the second incisor and first premolar. Examination of these roots showed no visible areas of absorption whatever, though the latter appeared to be twisted. On inspecting the radiograph of the canine it was at once apparent that there was a loss of substance—i.e., enamel and dentine—at the summit of the cusp. It was possible to trace a direct line of continuity between the cystic contents outside the tooth



FIG. 5.

Subcapsular odontocoele, as viewed from *lingual* side. C., unerupted canine;
C.C., cyst cavity ($\times 1$).

and the pulp itself (fig. 5). As yet I had not ascertained that I had before me a most unique specimen.

On excising the cyst wall there was no venous hæmorrhage, as I had anticipated from the colour of the tumour. Instead, the cystic contents were deeply discoloured and almost black. It was this that had imparted the blue appearance by reflected light to the oral tissues, a phenomenon similar to that frequently witnessed in connexion with hydroceles of the *tunica vaginalis*. The tooth was removed and the cyst wall dissected out; granulation tissue soon formed, and at the end of ten months healing had taken place, a mere shallow depression on the surface of the jaw marking the site of the odontocoele.

The tooth was at once carefully tested for the presence or absence of Nasmyth's membrane. The inner layer was found, but no cellular

layer. It was then treated by the Koch-Weil method, and the section showed that the tip of the crown was defective and its growth had never been completed (fig. 6). There was no absorption of tissue. My delight on learning this knew no bounds! I had never seen, imagined, read of, or heard of such an anomalous defect in the growth of the hard parts which had led to the formation of a hollow, tube-shaped core occupying the vertical axis of the upper part of the tooth. It was truly a marvelous thing, as if Nature had made a mistake by failing to completely construct the free surface of the tooth, and, finding out her error, had determined to keep it concealed from the light of day—that is to say, to

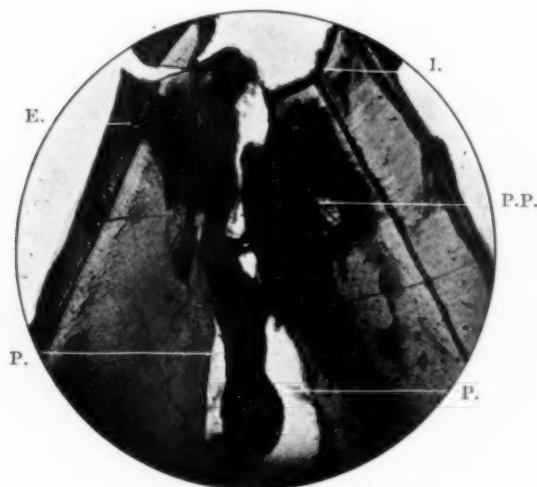


FIG. 6.

Longitudinal section of canine described in text. E., enamel; P., pulp cavity; P.P., pulp tissue extending laterally into dentine; I., massive edge of tooth ($\times 20$).

cause the tooth to remain buried in the bone! Had it erupted in the ordinary manner it is impossible to guess what the consequences would have been. These had better be left to the imagination of my readers! Soft tissue, composed of small cells, extended vertically into the pulp cavity, and also slightly laterally, in places, into the dentine. As the specimen was infinitely precious, I did not care to reduce its bulk too much, for fear of losing the parts, so I am unable to determine very

accurately the nature of the histological elements, but I believe them to be identical, or nearly identical, with those of the pulp, but profoundly altered, by contact with the cystic fluid, in shape and natural characteristics. The pulp itself was composed, at all events at its coronal portion, of broken-down cells, the odontoblasts were unrecognizable, the nerve bundles obliterated, while a few long endothelial-like cells closely applied in bundles represented the collapsed and shrunken walls of the vascular system. It is impossible for anyone to say what had happened to the enamel organ at that part which was going to form the summit of the crown. It could not have been due to septic disease of a deciduous predecessor, for one would then get the whole of the enamel organ deranged with regard to its usual functions. Apparently there was a sudden cessation of the work of the ameloblasts at that spot, leaving not only a



FIG. 7.

Canine (T.) imbedded in right superior maxilla, as described in text ($\times 1$)

breach of surface but a direct opening into the pulp cavity. After suitable preparation the cyst wall was microscopically examined. It was very thin, only measuring .25 to .75 mm. Composed of firm fibrous tissue it had a lining of numerous soft, large epithelial cells, held loosely together by a frail cementing substance. It was in no sense a compound epithelium, as seen sometimes in both dental cysts and follicular odontomes, nor were they secretory cells. It was, I believe, the external epithelium of the enamel organ swollen by absorption of cystic fluid, and the cyst wall was nothing more nor less than the dental capsule itself.

I have been unable to determine the conditions attaching to the maxillary canine. The patient is unwilling to have it removed, and can throw no light on the loss of the crown, which, as revealed by the radiograph, it had already sustained (fig. 7). To the accident of the presence

of a congenital lesion of the hard parts of the crown, and not to impaction or delayed eruption, can be attributed, I believe, the evolution of this cyst. Here we have, then, an example of a sub-capsular odontocèle—i.e., a cyst originating beneath the capsule or follicle of an abnormal tooth, as a result of the effusion of lymph from the neighbouring blood-vessels, into a potential cavity produced by a developmental defect of the summit of its crown. It may be suggested that the same morbid phenomena might have been induced by serous exudation into a space or spaces of a vacuolated follicle. But here the capsule had undergone no such retrogressive metamorphosis on account of its being retained in the jaw, as already mentioned. Hence the opinion that a cavity had existed through something unknown happening to the formative cells of the enamel organ many years ago, and effusion of lymph into the cavity thus created.

An Extra-capsular Odontocèle. (B) The case of Dr. C. N.

Interesting and instructive as is the case just detailed, the second one to which I wish now to direct your attention is even more remarkable. And it is further distinguished by the report of the subjective symptoms kindly furnished to me by the patient himself. To him, and to Professor J. H. Teacher, of the University of Glasgow, I am much indebted for permission to publish the facts.

Past history: The maxillary incisors and premolars were removed in 1899. One was broken, two were quite sound, and caries was present in the rest; the sound teeth were sacrificed for the purposes of giving firmer hold to a bridge which was to be constructed. The denture was worn for ten years. About three weeks before the tumour "became cystic"—to quote the patient's own words—he broke the plate through the middle, and he continued to wear it, being too busy to have it attended to. He believes that the movements of the fractured appliance irritated the jaw, and "stirred the latent tumour into activity." His dentist pointed out to him ten years ago that the permanent maxillary canine in the right side was missing, and had apparently never erupted.

Present history: "The patient's attention was first directed to a small enlargement of his cheek in January, 1909. He noticed it when washing his face, being conscious that the right cheek was fuller than the left. On feeling it more carefully he could make out a round, hard prominence close by, and at the same level as the right ala of the nose. His first impression was that a boil was forming, but it did not make

any progress in the way of 'ripening.' About two weeks later a little swelling appeared in the mouth in the angle between the upper lip and the gum, and in the region of the second incisor and canine teeth. This was fluctuant, and the patient thought that it probably contained pus. He incised it, but did not see pus, and in a few days it had attained the same size again. He then called in a brother practitioner, who incised it down to the bone, and it was then apparent that the swelling was a cyst. It soon filled up and became considerably larger, and the patient then thought it advisable to consult a surgeon with the view to its removal. Up to this point there was no pain associated with it beyond a throbbing in the gum and part of the hard palate."

"About March 18 the patient saw one of the leading operating surgeons in Glasgow, who examined the cyst and advised its removal under cocaine and adrenalin. A few days later he proceeded to do this, and on dissecting off about a third of the cyst wall he found it firmly rooted to the bone of the upper jaw. He sheared off the dissected portion of the cyst close to the bone with the view to its pathological examination and further operative treatment. From the clinical appearance of the tissues he was of the opinion that the swelling was malignant in character. Examination was made of the detached portion, and on the patient's return two days later, he was informed that the tumour was of the nature of a myeloid sarcoma. This diagnosis, however, was disputed by the pathologist who, as a personal friend of the patient, was called in to give an opinion. He was very emphatic that there were no tissue elements to warrant a diagnosis of malignant disease, and it is only fair to the operating surgeon to say that he abandoned the diagnosis of myeloid sarcoma, and consented to perform a much more modified operation than he originally intended, although still feeling that there was a malignant element in the case. The operation decided upon was to remove the affected area with a margin of healthy tissue around it. This was done on March 24, a wedge-shaped portion of the upper jaw being removed, and a considerable part of the bone of the anterior wall of the antrum included, the membrane being left. The cavity in the mouth healed up without any difficulty, although the antrum became affected and necessitated douching through the nose. Eventually the wound closed completely, and the mouth has remained sound and well after a period of twelve months. The wedge-shaped portion of bone removed has been examined by Mr. A. Hopewell-Smith, who has expressed the opinion that the tumour was of a perfectly simple kind." (Patient's report.)

Appearance of the tumour: The specimen consisted of a V-shaped piece of tissue bounded internally by the median line of the palate, and externally by a line running backwards and inwards through the premolar region (fig. 8). Its width in front was 3.5 cm. The labial aspect showed an opening in the alveolar process 22.5 mm. long, 9 mm. wide, the lower margin being placed 6 to 9 mm. from the free edge of the alveolar process. It was made by the incision for the evacuation of the cystic fluid. At its base, and at a distance of about 4 mm.

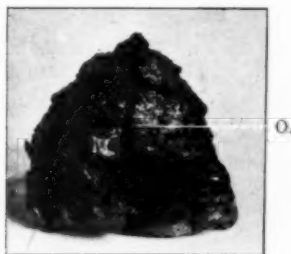


FIG. 8.

Labial aspect of soft tissues of right superior maxilla, to show horizontal incision to evacuate contents of cyst ($\times 1$).



FIG. 9.

Extra-capsular odontocoele, as viewed from *mesial* aspect; incision made to right of line marked O. in fig. 8. P., palatal surface; L., labial surface, where fluid accumulated; C., canine; C.M., calcified mass ($\times 1$).

from the surface, lay a calcified mass (fig. 9). The main object in the preparation was a canine fully formed and well developed, 25 mm. in length. Its crown was almost entirely embedded in a solid mass of brittle, calcified material, which was yellowish in colour and partly translucent by reflected light. During the digital disturbances conse-

quent on the operation this calcified cap had obviously been dislodged from the position it had occupied for many years and had become detached, and the crown of the tooth was shelled out from it with the exception of a small excrescence, which, still adhering to its distal aspect, gave the clue to the pathological conditions which had been occurring (fig. 10). The nodule measured 4 mm. in length, the dimensions of the hard cap being 14 mm. by 12 mm. There were no marks of absorption on the surface of the enamel (fig. 11). Nasmyth's membrane was present. I succeeded in obtaining fragments of its cellular layer. The calcified mass was obviously the dental capsule which had undergone a retrogressive metamorphosis. It had not been completely infiltrated, as portions still retained their fibrous characters (fig. 12). Pathological calcification or petrification occurs almost without exception only in degenerating, dying, or dead tissue as



FIG. 10.

Tooth everted from its surroundings to show calcified nodule (N.) attached to surface of crown ($\times 1$).

Hektoen and Riesman [5] record (vol. i, p. 113). Thus its appearance can probably be explained by the fact that the dental capsule of the unerupted canine had died, and formed a nidus for the lime-salt infiltration, thus differing very greatly from the follicle in the preceding sub-capsular odontocoele. The composition of the hard mass was found to be calcium carbonate largely. On placing a fragment under the microscope, and allowing a weak solution of hydrochloric acid to run, by capillary attraction, beneath the cover glass, bubbles of gas were immediately evolved, the residue of organic material presenting an amorphous structure with a tendency to the formation of a pattern of spherules. Around the surface of this calcified capsule was developed the cyst, and what was apparently the "bone of the jaw" mentioned in the patient's notes was its exposed surface. Histologically examined, the soft parts

consisted chiefly of connective-tissue fibres with numerous small cells, changed connective-tissue corpuscles, tiny hæmorrhages, clusters of fat cells, blood-vessels, and a small amount of bone which represented all the remains of the thinned and expanded external alveolar plate. The cyst wall was lined with epithelium. There were no microscopic signs of inflammation.

Whence came the cystic fluid? It is difficult to determine. Eliminating every possible structural element in the gum which could give rise to a cyst, it might be conjectured that the cells of the walls of certain lymph spaces in the submucous tissues had broken down, and

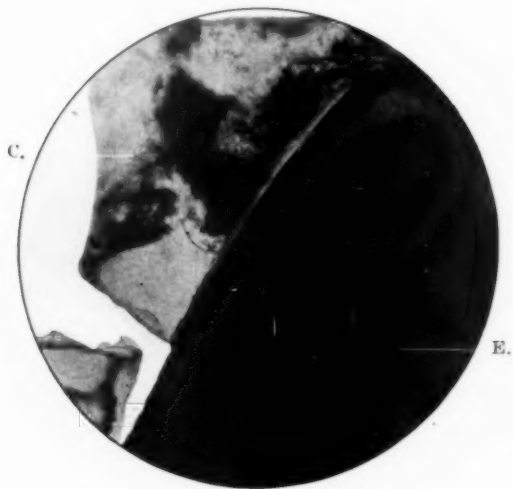


FIG. 11.

Photomicrograph of crown of tooth described in text. E., enamel;
C., calcified nodule ($\times 45$).

produced, by rapid multiplication, the cyst, through irritation from the friction of a loose, fractured mechanical appliance on the one side, and the hard unyielding surface of the ossified capsule on the other.

At first sight I regarded the dental condition as a calcified follicular odontome. But, on comparing it with the specimens in the museum of the Royal College of Surgeons, and especially with one presented by the late Samuel Cartwright, I decided that this was not so. For the cystic fluid was *outside*, not *inside*, the capsule, which was not expanded,

but remained, in spite of its petrification, closely adherent to the surface of the tooth. Hence I venture to call it an extra-capsular odontocoele, and to explain its origin from the breaking down of epithelium or endothelium in the soft tissues of the jaw lying between, and stimulated into growth by the pressure, and perhaps friction, of two hard, unyielding substances.

There are some further problems surrounding this case which can never possibly be solved: The age of the patient when calcification of the capsule took place; the possibility or otherwise that this calcification, acting as an obstruction, was the cause of the non-eruption of the tooth; the histological characteristics of the actual structures in the soft superimposed parts, which, on breaking down, produced the odontocoele; the date of its formation; and the nature of the still more mysterious forces which governed the impregnation with lime salts of the dead or dying follicle.



FIG. 12.

Same as fig. 9; its *distal* surface. D.C., dental capsule; T., canine ($\times 1$).

The fact that mechanical agencies play a not inconsiderable part in the production of disease calls for no special comment. And teeth which undergo moliminous eruption, or do not erupt at all, as is so frequently the case with the third molars, may act as foreign bodies and produce inflammation and absorption of the surrounding parts, and even give rise to cystic tumours of the upper jaw.

Cystic Adenomata.

To illustrate this, I show, but do not now describe, a cystic adenoma of the left superior maxilla, which has been occasioned entirely by the mal-position of the third molar and its fruitless efforts at eruption—an

adenoma which, filling up the greater portion of the antrum in its upper part, has on its inferior aspect induced, on breaking down, an ulceration of the soft tissues of the hard palate in the molar region.

CONCLUSION.

I think I have fairly substantiated my reasons for the introduction of a new term in dental pathology, and have established that neither case accords with the definition of a follicular odontome, though bearing some superficial resemblance to one, as in neither instance was the *locus principii* of the cysts to be found in the stellate reticulum of the enamel organ, nor did their contents approximate in the very least to those usually found in such.

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(The discussion on this paper was adjourned until the June meeting.)

Odontological Section.

May 23, 1910.

MR. WILLIAM HERN, President of the Section, in the Chair.

Report of the Honorary Curator.

By J. F. COLYER, M.R.C.S., L.D.S.

I HAVE to report that the transference of the Museum from 20, Hanover Square, to the Royal College of Surgeons was carried out during the autumn of last year. Since then the greater part of the time has been occupied in rearranging the specimens in their new home. I have pleasure in stating that the Museum is now for all practical purposes sufficiently straight to allow of the specimens being used for research purposes.

It is intended during the coming year to re-catalogue the specimens, and by this time next year I hope to be able to report that this has been carried out.

There are several sections of the collection which need extending, and I would appeal to Members of the Society to assist by giving specimens towards these sections. I shall be delighted to receive specimens of any kind, but more especially of odontomes, anomalous shaped teeth, and skulls illustrating irregularities of the teeth.

During the coming year it is intended to rearrange the section dealing with irregularities in position of the teeth, and considerably extend this section, bringing it as far as possible thoroughly up to date.

The number of specimens added to the collection during the past year is small. Amongst those to be noted are :—

Specimens presented by Mr. Hopewell-Smith, illustrating a case of infective disease of the jaw.

A skull of a black American bear, by Lord Walsingham.

Two wild rabbit skulls showing extra incisors, from Professor Beard.

An agnathic sheep.

Skull of wild boar showing actinomycosis.

Skull of a calf showing defective maxilla.

The Spindles of von Ebner.

By DOUGLAS E. CAUSH, L.D.S.

THE study of the spindles of von Ebner is exceedingly interesting from the fact that their position in the teeth is as unique as it is exceptional, they being developed from a mesoblastic tissue (the dentine) and found situated in an epiblastic one (the enamel). Various theories have been propounded as to how the interchange of tissue has taken place, and to introduce this subject for your consideration is the object of my paper.

The interest in these spindles is intensified by the fact that their existence is denied by some writers, as Waldeyer and Hertz; while others, including von Ebner, Tomes, Wild, and Holländer, acknowledge their existence but are not agreed as to whether they are pathological or normal in their development. If any here have doubts as to the existence of these spindles, they can easily set these doubts at rest by the following experiment: Take a bicuspid tooth and immediately after extraction place it in an alcoholic stain of gentian violet for two or three months, then cut a section from the tooth through the cusps, and in the majority of cases the result will be similar to that seen in Slide 1.

POSITION.

The position of these spindles varies somewhat in the different teeth. They are usually to be found in the greatest numbers in the first-formed enamel of the tooth. Thus, in the incisors and canines (if found at all)

they are to be seen in the enamel immediately over the first-formed dentine (Slide 2) ; the spindles are also found, though not so frequently, on the sides of the dentine (Slide 3) ; passing from the apex of the dentine towards the gum margin they are less in number, and it is very unusual to find them near the termination of the enamel. In the bicuspid (Slide 4) the spindles are to be found in the greatest number on the outer or first-formed cusp of the dentine ; sometimes they may be found on the inner cusp of the tooth, but it is unusual to find them in the enamel near the gum margin, and most unusual to find them on the dentine between the two cusps. In the molar it is usual to find the spindles in the enamel of all the cusps, but not so frequently in the enamel between the cusps ; in this position (the enamel between the cusps), both in the bicuspid and the molar, there appears what I take to be a modification of these spindles, bundles of the tubuli of the dentine passing into the enamel (Slide 5).

DEVELOPMENT.

The difficulty of studying the development of these spindles is very great owing to the fact that in the temporary teeth they are small in size and, as a rule, few in number (Slides 6 and 7). Nor does the study of the development of the teeth of animals help to elucidate the problem, as I have not found any spindles in the teeth of the animals I have examined except in the gorilla, where they are small in size and few in number.

The examination of sections from the human embryo helps to elucidate some of the difficulties of the mixing of the two tissues (Slides 8 and 9). These slides from the incisor and premolar region of the embryo show that it is not at all unusual to find at the junction of the two tissues, the point where the spindles are the most pronounced in the permanent teeth, that at the commencement of the development of the enamel and dentine, the line of demarcation is by no means pronounced, and there is frequently a mixture of the epiblastic and mesoblastic tissue. As the development proceeds this line of demarcation becomes more pronounced, and the organs producing the enamel and the dentine appear to settle down to their work more perfectly. The result of this interchange of the two tissues is seen in many sections of the permanent teeth (Slide 10). In these teeth the first-formed tissue does not appear to take the character of a normal granular layer, but it appears as if portions of the mesoblastic tissue have been

forced into the epiblast, and the latter tissue has been calcified around the former (Slides 11 and 12). If this is the case, it is not difficult to follow the development of the spindles.

I do not think the hypothesis of Walkhoff is correct when he states there has been an absorption, or resorption, of the first-formed dentine to assist in the formation of these spindles. As far as I have been able to discover, there are no cells in the epiblast that are capable of absorbing the dentine after it is once formed, and the development of the enamel being simultaneous with that of the dentine would quite prevent such absorption taking place. He suggests, as a proof of this hypothesis, that there is little or no branching of the tubuli at this point owing to the absorption. My slides will not bear this interpretation, as the branching of the tubuli is decidedly more pronounced where the spindles are developed than it is where they are absent (Slide 13).

Though the spindles are less in number and smaller in size in the temporary teeth (Slide 14), there is, I think, to be found in these teeth proof that they have certain functions (of which I shall treat later on) to perform. With the absence of the spindles there is an increase in the number of the ends of the tubuli that pass into the enamel. This is very pronounced in the temporary teeth (Slides 15, 16, and 17), and tends, I think, to prove the method of development I have suggested.

SIZE.

In size these spindles vary considerably, but much more so in the permanent than in the temporary. The smallest spindle I have measured in a permanent tooth was 6μ by 2μ , and they are to be found from this size to that of the largest measured, which was 70μ by 4μ . The greater increase of length over width is, I think, another proof of the theory of the development I have suggested.

SHAPE.

In shape they vary quite as much as they do in size, and are to be found spindle-shaped, fusiform, oval, beaded, and sometimes, though rarely, branched. However much they vary in their shape, they are usually the same width at their base as the tubuli of which they are a continuation. The shape is, I think, controlled by the power the matter forming the spindle has of resisting the pressure of the enamel cells during the process of calcification.

CONTENTS.

Their contents are, I believe, protoplasmic in character and similar to the central portion of the tubuli of the dentine. Owing to the difficulty of obtaining the contents of these spindles free from the surrounding tissue, I have not been able to make an examination of it. At the same time, as these spindles stain in a similar manner to, and are a continuation of, the central portion of the tubuli of the dentine, it is not unreasonable to assume that they are of the same composition as the contents of the tubuli, though they differ from the former in that they appear to have no sheath.

RÖMER'S CORPUSCLES.

Occasionally there is to be found in these spindles portions that do not take the stain as readily as the rest of the contents; they are circular or oval in shape, and are known as Römer's corpuscles (Slide 18). Römer suggests that they are a peculiar form of nerve-ending. I do not think this probable, as there is no record of the nerves being found in the tubuli; and if these bodies were, as he suggests, nerve-endings, there would certainly have been found some connexion between them and the nerve-supply of the pulp. It appears to me that these so-called corpuscles are dead, or dying, cells in the mass of protoplasmic tissue that forms the spindle, as in teeth that have been stained for but a short time, and in sections that have been surface-stained, they stain but slightly; but in teeth that have been stained thoroughly these corpuscles are also stained, and it is difficult to find any of them.

AIR SPACES.

In teeth that are allowed to dry, so that the contents of these spindles are allowed to contract (Slide 19), it is not at all unusual to find air enclosed within the spindles; but in teeth that are kept moist until the sections are made, it is very unusual to find any air enclosed in the spindles.

NORMAL OR PATHOLOGICAL.

The conclusion I have arrived at, after the examination of a large number of teeth, is that these spindles are normal, and not pathological,

and have certain functions to perform in the life of the tooth in which they are found. An analysis of the enamel sections in my cabinet tends to confirm this view; teeth of all kinds have been examined, and the following table shows the percentage:—

Temporary teeth	50.0 per cent.
Permanent teeth	88.9 "
Incisors	85.4 "
Canines	72.2 "
Bicuspid	100.0 "
Molars	98.0 "

It is not at all unusual to find perfectly calcified teeth containing a large number of spindles, whilst in teeth that are badly calcified, or teeth in which the enamel is abnormal, the spindles are often absent (Slides 21, 22, 23, and 24); in the four slides shown of teeth with abnormal enamel, only one contained spindles. These spindles are to be found in the teeth of the ancient as well as in those of the present day; I have found them in the tooth of the Saxon kindly given me by Mr. Walter Harrison, and in a tooth of ancient date found in Cornwall and kindly supplied by Mr. C. Robbins (Slide 25). From the slides I have examined it would appear as if the teeth of modern times contained spindles that are larger in size and much more numerous than the teeth of the earlier period.

These spindles play a very important part in caries of the enamel, especially in connexion with that Miller describes as the secondary decay of the enamel. I regret it is impossible in this paper to enter fully into the part they play in the work of destruction, owing to the time at my disposal, and to the fact that, to make myself clear, it would be necessary to consider portions of the tooth tissue outside my subject; but think it will interest you if I show on the screen some slides (26, 27, 28 and 29) showing bacteria in these structures under consideration, the spindles of von Ebner.

FUNCTIONS.

In conclusion, I should like briefly to bring before you what I believe to be some of the functions of these spindles, with the object of obtaining your views upon this important, but rarely-discussed subject. If expansion and contraction takes place in the enamel, as I believe it does, owing to the difference in the temperature of the foods taken, may not these spindles be a provision to allow for that expansion or

contraction without the fracturing of the hard and dense tissue of which the enamel is composed? It is in man alone we get the variation in the temperature of the food taken, and it is in man these spindles are found. Again, in the teeth of modern man the spindles appear to be larger and more numerous than in those of our ancestors. May not the food of to-day have something to do with this increase both in the size and number, especially as these spindles are to be found in that portion of the tooth where the results of either expansion or contraction would be most disastrous to the organ in question?

SENSATION.

It is a recognized fact that the sensation produced by heat or cold passes through the enamel. In all teeth where the pulp is alive there must be, of necessity, some means whereby this so-called sensation can be gathered up to convey it to the pulp through the tubuli of the dentine; and what is more probable than that these spindles, which are in direct communication with the tubuli, are one of the means of conveying this sensation to the pulp, either as a means of warning, or to incite the pulp to self-protection in the formation of secondary dentine? It may be only a coincidence, but we certainly find secondary dentine more frequently formed in that portion of the pulp-chamber corresponding to the position of the spindles than in any other part of the chamber or canals.

DISCUSSION.

The PRESIDENT (Mr. Hern) said Mr. Caush had written an interesting paper on a somewhat abstruse subject. It had been a moot point with some histologists as to whether these spindles were pathological, anatomical, or produced by the methods of preparation for the microscope, but he thought Mr. Caush had shown conclusively that they were very real anatomical structures, and he had given some reasons for their occurrence—viz., that their presence might help to prevent fractures of the enamel when under the influences of thermal changes, or they might be concerned with the question of sensation. It had struck him that their function was perhaps very similar to that of the pieces of hoop iron which were used in buildings to tie walls together—i.e., their office might be to prevent the enamel stripping away from the dentine. At any rate, as a mechanical binding they seemed to him to serve an obviously useful purpose.

Mr. D. P. GABELL felt some hesitation in criticizing a paper by Mr. Caush, seeing that his very earliest attempt at microscopic work was done under Mr. Caush's tuition. The suggestion that the spindles of von Ebner were due to the expansion and contraction of enamel seemed to have one disadvantage—namely, that if there were contraction and expansion of the enamel it would seem that that expansion and contraction would be greatest at the surface of the enamel and not at the deeper parts, and therefore the spindles would be found near the surface, instead of only at the junction of the dentine and enamel. If they were due to expansion and contraction, was it not more probably expansion and contraction of the dentine such as was obtained when drying a specimen? He could only suggest to Mr. Caush as one method of deciding that point that he should examine some unerupted teeth. If the specimens were kept moist it would exclude the question of the drying of the dentine causing the spaces. Some of the specimens exhibited appeared to show the mingling of the dentine and enamel, and he would like to know whether Mr. Caush could safely exclude those sections from the class of sections where the junction of enamel and dentine was cut obliquely, a thing that was likely to happen near the apex of a tooth, showing one tissue behind the other. That could not be told from the photographs, but only from an examination of the specimens with the finger on the fine adjustment.

Mr. LEWIN PAYNE said the members would be grateful to Mr. Caush for once again adding another valuable contribution to the histology of dental structures. One suggestion that had occurred to him with regard to the use or possible function of the spindles of von Ebner was that the position in which they occurred might lead one to imagine that they were there for the purpose of adding strength to the tooth. They were found especially over the masticating edge of the tooth, extending towards the masticating edge, whereas in other parts of the dentine and enamel they were practically non-existent. One would imagine that they really served the function of helping to strengthen the enamel, quite independent of the question of the action of heat in causing expansion and contraction. With regard to the question of expansion and contraction, it might be imagined that if the function were to preserve against such expansion and contraction the spindles would extend all round the edge of the dentine. With regard to their origin, he thought Mr. Caush had very clearly shown that they arose from the dentine itself and not from the enamel, that they were really derived from the dental papillæ, and that the enamel was calcified around them. Possibly they might explain some of the sensitiveness of the enamel, which was difficult of explanation without considering that the enamel contained organic matter.

Mr. E. B. DOWSETT said the spindles of von Ebner had always interested him, principally with regard to their function. He agreed with Mr. Gabell's suggestion with regard to the mingling of the dentine and the enamel, and probably the appearance was due to the thickness of the section as well as the obliquity of the cutting. As regarded the origin, in hard sections one frequently

observed the spindles where there was a very definite line of demarcation between the enamel and dentine, and therefore it was not a true mingling. It seemed to him that where one saw a definite spindle coming off from a very straight line of demarcation it must be due not to a mingling but to the growth of an odontoblast out between the ameloblasts. There was no definite membrane between the two, and it seemed to him quite easy for the process of an odontoblast to grow out upwards between the ameloblasts, and the enamel would be formed around the process of the odontoblast. With regard to function, the only point that seemed to him really probable was the function of sensation. The spindles were found at places where the enamel was mostly worn—the tips of the cusps—and therefore spindles in that situation would be irritated by the attrition of the points of the cusps, and sensation would be conducted to the cusps and secondary dentine laid down as suggested. That seemed a very good reason why they were there—to convey sensation to the pulp and thereby lay down secondary dentine for protection.

Mr. H. LLOYD WILLIAMS said that, although his ignorance of microscopic work was great, he thought the mixing of enamel and dentine to the extent that Mr. Caush's thesis demanded required very clear demonstration indeed. With regard to the function of the spindles, the position in which they were placed, on a very sharp curve and always under the cusps, would bear out the idea which Mr. Lewin Payne had suggested, that they were there on account of the pressure exercised on the cusps. As a matter of physics their direction was correct to take off the direct pressure of mastication. The little slits in the enamel would rather suggest they were there for that purpose. The enamel was placed over a very steep curve, and, being extremely solidly calcified, would be very likely to split off far more frequently but for the fact that Mr. Caush had so clearly demonstrated. He thought there was an objection to the idea of their function being to avoid the splitting of enamel on contraction and expansion from changes of temperature. If they were there for that purpose, and supposing they contained living protoplasm, the teeth would cause a great deal more pain than they did from the immediate changes of heat and cold, in the same way that they suffered when enamel was chipped off and dentine exposed.

Mr. CAUSH, in reply, said he had anticipated being treated much more severely. With regard to the remarks of the President as to the use of the spindles, he quite agreed with him, but thought the tubuli of the dentine that passed through into the enamel and were not developed into spindles played a still greater part. That was noticeable in the fact that in the temporary teeth, where the spindles were few in number, the tubuli passed into the enamel much more freely than in the permanent teeth. In this way the two tissues were very much helped to hold together, and that fact would assist in solving one of the difficulties histologists had found. He sympathized with Mr. Gabell, who was a microscopist, and knew the difficulty of cutting exactly in the plane required. In his own case the way in which the teeth were stained

was of assistance, as the staining showed the difference between the two tissues, and enabled him to see how the grinding was progressing. In some cases where great care was not taken it might be that Mr. Gabell's observation set forth one of the causes, but if trouble were taken carefully to grind down the teeth the spindles would be obtained in many places where the plane was as near as possible parallel to that of the tubuli of the dentine. With regard to unerupted teeth, the first slide shown on the screen was from an unerupted tooth supplied by the late Mr. John Ackery. Mr. Ackery was exceedingly kind in sending him a supply of unerupted teeth that he had removed for the purpose of regulation. [Mr. GABELL asked whether the teeth were kept wet.] Mr. Caush said they were placed in alcohol and sent down to him immediately after extraction, and he was very careful not to allow them to get dry. He did not mean to imply in his paper that expansion and contraction caused the spindles, but rather that the spindles were there to prevent trouble to the enamel as the result of expansion and contraction. With regard to the suggestion that the spindles were there for the purpose of receiving the sensation of heat and cold, consideration should be given to the fact that when a hot or cold fluid was put into the mouth it did not run all round the teeth. He did not say it was the function, but a function, and he thought there was a certain amount of ground for the theory.

Odontological Section.

June 27, 1910.

Mr. C. F. RILOT, Vice-President of the Section, in the Chair.

Some Egyptian Skulls.

By ARTHUR S. UNDERWOOD, M.R.C.S., L.D.S.

MR. ARTHUR S. UNDERWOOD exhibited a number of Egyptian skulls in which the front teeth of both jaws had the appearance of mutilation. At the College of Surgeons, the British Museum, and other places, he had looked through 150 Egyptian skulls, and out of them 125 showed apparent mutilation of the front teeth, a large amount of the substance having been removed as would seem deliberately or from injury. He had found the mutilation solely amongst Egyptian skulls, with the exception of one or two Peruvian. There appeared to be three possible explanations: the damage might have occurred from a rude attempt to cure painful teeth, the teeth might have been filed for a religious or tribal purpose, or the injury might have been post mortem, caused by splitting from dryness due to burial in the sand. The skulls might be anything between four thousand and six thousand years old, and it was quite possible the teeth had become friable and fragile, and thus liable to injury. But that did not seem to him to explain why only the front teeth were injured. It almost appeared as if the damage had been caused for the purpose of an inlay. There were two or three skulls where the front surface had been removed from the upper incisors somewhat similar to the deliberate mutilation in the case of some Negro skulls. At the Royal College of Surgeons' Museum there were a certain number of mummy heads from which the integument had never been

removed, and it would be interesting to see the condition of those teeth in order to form an idea as to whether the injury was due to the sand and to the rattling of the dry bones when in process of removal. One skull, that of a Babylonian woman aged about 40, showed no kind of splitting or change, and that skull was preserved in a sarcophagus under practically the same conditions as the majority of the Egyptians. There was an ancient skull in the British Museum, dating from the Fifth Dynasty, which showed no mutilation of the teeth.

DISCUSSION.

Mr. F. J. BENNETT suggested that a ready means of discovering whether the injuries were post mortem or ante mortem would be to make a section of a tooth and see whether there was secondary dentine in the pulp cavity. If the teeth had been filed during life and had not reached the pulp cavity, there would certainly be a deposit of secondary dentine to make up for the deficiency due to filing; whereas, if they had been filed through to the living pulp and the pulp had died there would be a deficiency of dentine compared with the back teeth which had not been filed.

Mr. HOPEWELL-SMITH said that some years ago he had an opportunity of unravelling the skull of a mummy that had been brought to England from Cairo. It was supposed to be four thousand years old. The jaws were carefully unwrapped, and an examination showed the existence of no caries and no marks of mutilation; and the wisdom teeth were well formed, all the teeth being quite sound. Recently a friend had brought from Khartoum the skull and the lower jaw of two Dervishes killed at Omdurman. The bones had been lying on the battle-field covered with sand. The enamel had split off several of the teeth, mainly in front of the mouth, and it was thought that that was probably due to the extremes of temperature in the climate of Egypt. The contraction and expansion of the dentine in a variation of temperature which amounted to forty degrees would, in time, produce that effect upon skulls lightly hidden in the sands of Egypt. The skull was in the Museum of the Royal Dental Hospital. He would make an investigation as suggested by Mr. Bennett.

Mr. W. RUSHTON said some Egyptian skulls of extreme antiquity had been recently placed in the Museum of the Royal College of Surgeons, evidently skulls of the common people, as they were buried in the sand. The amount of destruction in tooth tissue was remarkable, and seemed to be too irregular to be of the nature of attrition or abrasion; it was probably post mortem. If Mr. Underwood took an X-ray photograph of the mummy head mentioned, he must not be surprised if he found the teeth were as perfect as some of those of the royal Egyptians in the Royal College of Surgeons' Museum.

Mr. HOPEWELL-SMITH said another skull in his possession had been brought from German East Africa and showed marks of mutilation, the two lower central incisors having been chiselled out. The young males of the Wagogo tribe were subjected to this operation, partly as a tribal mark, and also probably as a religious observance and in case of possible lockjaw.

Mr. GABELL said in some of the skulls shown by Mr. Underwood the pulp was freely exposed and there were no signs of alveolar trouble at the roots of the teeth, also the cuts were very clear and showed no rounding by wear. That led him to suppose it was a post-mortem injury.

Mr. ASHLEY DENSHAM said it was clearly stated by such authorities as Virchow and Schmidt that in no single instance was there any evidence of surgical treatment of teeth amongst the Egyptians. There was constant mention of excessive attrition and comparative immunity from caries, but no reference to mutilation, either artificial or natural. That rather led him to think that perhaps the skulls examined by Mr. Underwood were mostly those of the lower classes, who were buried in sand and not in sarcophagi. In the sand naturally the bones were subjected to more radical changes of temperature and extraction of moisture than those buried in sarcophagi. In the "Ebers papyri" several diseases of teeth and gums were mentioned and treatment suggested, but nothing was said about surgical treatment. The casual examination he had made of the skulls exhibited that evening led him to think that the damage was largely post mortem, and he had noticed that in the back teeth considerable portions of tooth structure had been split off, some apparently as recently as within the last six months. Although one could not rely too much upon the examination by expert Egyptologists who were not expert dentists, yet it was important to note that they had come to the conclusion that nothing in the way of filing of the teeth had been found in any single instance.

Mr. UNDERWOOD, in reply, said the difficulty of cutting sections was that the specimens were not available for the purpose. There were a good many alveolar abscesses amongst the teeth, but he could not say that they were due to injury. Mr. Hopewell-Smith had given him some assistance by showing that there was no change in the mummy, but there was in the Dervish. With regard to Mr. Densham's remarks, he had no great reverence for the opinion of Egyptologists on matters outside their own business, and the absence of reference in a papyrus of a habit was, after all, only negative evidence. He gathered from what had been said that it was the opinion that the changes were post mortem. After having considered Mr. Hopewell-Smith's cases and taken the X-ray photograph, he should be pleased to give some further information to the Section.

Case showing the Result of Extraction of the Six-year-old Molars.

By GEORGE THOMSON, L.D.S.

THE case I wish to show you is not singular but typical, and not one of the worst but one of the best. Eleven years ago, when the patient was aged 13, I extracted the four six-year molars and, incidentally, the first left lower incisor. Before showing the slides I wish to express my own views concerning this treatment. If they are wrong it does not much matter to you, for in the keen atmosphere of this Section they will not survive. I have surrounded the roots of one molar extracted with plaster of Paris, which may represent the bone in which this tooth was implanted. If the patient has lost four times this quantity of bone from his face, his features and expression must be marred considerably. You will see the face in profile. Correct occlusion has been made impossible, as the models will show. Professor Hill, in his book "*The Body at Work*," says: "The teeth are thirty-two in number, starting from the middle line of either jaw; the first two are incisors, with chisel-shaped, cutting edges. If they meet as they ought to do, *their edges are ground flat*." The incisors in this case overlap considerably, so that the rotary movements in mastication are prevented and the cusps are not worn down.

What I consider to be correct occlusion is seen in the Australian aboriginal, a pure type and genetically related to us, he being a dark Caucasian. His incisor teeth are said to present, in the adult, no peculiarity to differentiate them from his other teeth, because they are all worn down equally. One wonders whether the difference in our occlusion is so much racial and inherited as environmental and nutritional. It is pointed out by Dr. Ramsay, of Adelaide, that in the development of the molar teeth of the Australian the coronal surface of the molars face distally, whereas in the white races they face mesially. The extraction of the first molar must tend to further increase this fault.

Further, if teeth are extracted, the mesiodistal relation of the teeth is disturbed, and interstitial caries is most likely to result. Crowding of teeth does not cause caries. Again, the extraction of a molar tooth creates a nidus for pyorrhœa alveolaris, and pockets are formed quite early in life.

I have only touched the fringe of this subject in the short time at my disposal.

DISCUSSION.

The CHAIRMAN (Mr. C. F. Rilot) asked whether the molars were carious when extracted.

Mr. THOMSON said they were, but he did not think that affected the case.

Mr. RUSHTON asked whether the teeth at present were free from caries.

Mr. THOMSON said there was interstitial caries in two places.

Mr. RUSHTON thought Mr. Thomson had done the patient a very good turn in taking out the four teeth. From the models it would appear as if the patient had been a mouth-breather, as the jaw was somewhat narrow. The results of extraction were a slight loss of masticating power and a place left that should be carefully kept clean, but on the whole he approved of Mr. Thomson's treatment, as the wisdom teeth had had room to come forward and were quite functional. The teeth were regular and nearly free from caries. The patient's appearance was good, the somewhat narrow face being the result of mouth-breathing and not of extraction.

Mr. ROBBINS wished to know whether Mr. Thomson looked upon the extraction as a great mistake and gave it as an instance of wrong-doing. He himself, instead of calling it a doubtful operation, considered that in certain cases the removal of four sound teeth was the grandest operation that could be performed. There were many reasons why he should not select the six-year-old molars in thinning out, preferring the second bicuspid; but to say that on no account must the mouth be thinned out was absolutely against common-sense. He did not think it was possible to agree with Mr. Thomson that crowded teeth were not conducive to interstitial caries; his own impression was that it certainly did increase the chances of interstitial caries. The late Mr. A. H. Woodhouse held that in many cases where it was desirable to avoid apparatus and obtain a good result the second bicuspid should be extracted on either side, claiming that that relieved the jaws equally. The second bicuspid being midway between the symphysis and the tuberosity the regulation went on naturally backwards and forwards, and there was room ultimately for the wisdom teeth. In very many cases where he himself had performed the operation the mouths were perfect, with no interstitial decay, and the wisdom teeth in their places. Mr. Robbins was willing at any time to show a series of models to prove this point.

Mr. GEORGE THOMSON thanked the speakers for their criticisms of his case. Their congratulations on its success no doubt were intended as a heavy blow to his argument. The chief point in this case was that of defective facial contour. His attention was first called to this by observation of the face of a lady whose features were lacking in character, due as he believed to the extraction of the four first molars. He was glad this case was that of a boy, for it still more emphasized the importance of the lower part of the face in expression, and who could set value on the expression of the human face?

Discussion on Mr. Hopewell-Smith's paper on "Two
Odontoceles and some other Cysts."¹

Mr. F. J. BENNETT, said the subject of cysts of the jaw had formed a conspicuous part of the *Transactions* of the Odontological Society, but had been usually associated with the work of general surgeons. He thought Mr. Hopewell-Smith's paper was a prelude to a new departure. With the introduction of X-rays there were likely to be fewer communications from general surgeons and more from dental surgeons. To approach the treatment of a tumour of the jaw without the assistance of the X-rays required a very large diagnostic experience, and he would be a bold dentist who would run the risk of dealing with a sarcoma under the belief that he had tooth-tumour; but with the X-rays the diagnosis was reduced almost to a certainty, and a dentist might be as much at home in removing a tooth-tumour as the general surgeon. The paper Mr. Hopewell-Smith had brought forward was to introduce two new forms of cysts—termed "odontoceles"—and he further reviewed the classification of cysts generally, with the view of showing that the two particular cases mentioned did not fall within the usual classifications. He was disposed to dispute that, especially in the case which Mr. Hopewell-Smith called the extra-capsular odontocèle. It seemed to him that it should be called a typical follicular odontome. With regard to the subcapsular odontocèle, the case was an extremely remarkable one, and Mr. Hopewell-Smith might be forgiven for calling it unique. He thought in the paper there had been an unnecessary difficulty created by the introduction of such terms as "Nasmyth's membrane." He much preferred the simpler classification of those who treated it as the wall of the cyst. By keeping to the term "follicular wall," one knew exactly what one was dealing with, and if a tooth erupted covered by membrane it could be still called the follicular wall. Mr. Eve and Mr. Bland-Sutton made out that the fluid effusion came from the death and degeneration of the stellate reticulum, and it seemed to him there were two facts to be taken into consideration in that connexion. First of all, taking the whole volume of the stellate reticulum, and reducing it to a liquid state, would that represent a tenth part of the amount of fluid found in a cystic tumour of the nature described? If not, it was evident that the extra fluid must come from elsewhere. He did not believe the stellate reticulum died in that way; it might deteriorate to some degree and undergo some amount of liquefaction, but the main source of that fluid must come from the blood-vessels which supplied the stellate reticulum. In his experience the fluid of the cyst became apparent within a few weeks of

¹ Adjourned from April 25 (see p. 121).

the removal of the growth, and that seemed rather to point to some other source of the fluid than the stellate reticulum. If the tooth had a stellate reticulum for thirty years, and was in a deteriorating condition, it would surely have formed a large quantity of fluid which would become evident earlier. He hoped that Mr. Hopewell-Smith would succeed in getting the lower tooth he spoke of as being deficiently formed, as that might throw a great deal of light upon the subcapsular odontocoele.

Mr. WARWICK JAMES thought the conclusions drawn by Mr. Hopewell-Smith from the data were not justified. The tumours formerly would have been described as "dentigerous cysts," but they were now called "odontocoeles," with a further subdivision into subcapsular and extra-capsular. The introduction of those three names was based upon two cases only. Nor were cases previously described cited as examples. The term "odontocoele" was not defined, and as it did not form, in the author's opinion, a subdivision of a class already in existence, but a new class in itself, a definition was most necessary, and therefore a definition from Mr. Hopewell-Smith would be most valuable. In the first case, the relationship of the cyst to the tooth and the lining of epithelium of the cyst-wall were characteristic of a dentigerous cyst. It was true that the epithelial cells were large, but they were described as being derived from the external epithelium of the enamel organ, the view most people now held concerning the lining membrane of a dentigerous cyst. The epithelial character of this lining was largely based on Mr. Hopewell-Smith's work. The tooth itself was remarkable, and it was difficult to account for such a condition except from a lesion occurring at a very early period. The nature of the tooth did not necessarily affect the character of the tumour. It was well known that teeth in dentigerous cysts were usually perfect as far as they were developed, although most commonly incomplete, but it was well known that malformed teeth did occur in such cysts. In regard to the second case, the view that it was a dentigerous cyst could not be so readily held, but there was no other pathological condition which could otherwise explain the case unless it were a dental cyst associated with an unerupted canine. The early clinical history would suffice for either of those explanations, but when an incision had been made the tumour was often considerably altered by the inflammatory process which supervened, the epithelial lining often being destroyed. The subsequent history was obscured considerably by the calcification of the degenerated tissue, which frequently occurred in connexion with chronic forms of inflammation. In fact the subsequent history was such that it was hardly justifiable to accept the case as a new type of cyst in connexion with the teeth. Mr. Hopewell-Smith attached considerable importance to the fibrous tissue connected with the cysts and had endeavoured to associate it with the tooth follicle. In all innocent growths an adventitious capsule of fibrous tissue was formed as the tumour increased in size, the neighbouring fibro-connective tissue becoming

arranged round the growth to form a capsule. That, he thought, was the explanation of the function of fibrous tissue surrounding any cyst. With regard to the origin of the cystic fluid in dentigerous cysts, if the view put forward by Mr. Hopewell-Smith were accepted, the position was reached which was originally taken up by Mr. Tomes, when he compared such cysts to small cysts situated over erupting teeth. The external epithelium formed a cap over an erupting tooth and fluid accumulated beneath it. It was difficult to conceive that fluid passed from the surrounding tissues into the cystic space by a secretory process. It was more easily explained by a continual proliferation and degeneration of the lining epithelium such as occurred in a dental cyst.

Mr. GABELL thought it was a pity new terms were introduced into a subject without very careful consideration, and certainly when introduced they ought to have a very distinct meaning. The word "odontocoele," as simply implying a tooth-tumour, was identical with the word "odontome," and it was confusing to have two words with the same meaning. He could only suppose that Mr. Hopewell-Smith having taken so much trouble, and having done so much work, felt he had to add a title to it. For the great care and thoroughness with which Mr. Hopewell-Smith had investigated and recorded these and many other very interesting and valuable odontomes we were much indebted to him.

The CHAIRMAN thought all the members would be in sympathy with Mr. Gabell in being shy of the multiplication of names, especially when they were long and difficult to understand.

Mr. HOPEWELL-SMITH, in reply, said that in everything one was aiming to get at the real pathology of the conditions and to find out the truth. It was only possible to make deductions from what one had observed, either macroscopically or with the aid of the microscope, allowing for possible errors in the preparation of the specimens. With regard to the objection as to new terms, that was a matter to which he had given much consideration. He wanted to define something which, in his opinion, was not a dental cyst nor a dentigerous cyst. The early part of the paper led up to the two cases which were neither, in his opinion, dental cysts nor dentigerous cysts. A dental cyst had an inflammatory origin, and he came to the conclusion that neither of the cases was a dental cyst properly speaking. Both were tooth-bearing cysts in a sense; but it should be remembered there were three meanings to words—the literal meaning, the specified meaning, and the changed meaning. In favour of his argument, he referred to Liddell and Scott's lexicon, and quoted from Dunglison's "Dictionary of Medical Science," 1904, and Keating and Hamilton's "Pronouncing Dictionary of Medical Terms," for the reasons of calling each a tooth-tumour. He pointed out that the *literal* meaning of "adenocoele," for instance, was a "gland tumour," its *specified* meaning a cystic adenoma; the *literal* meaning of bubonocoele, "a swelling in the groin," its *specified* meaning an

inguinal hernia; and varicocele, which meant the twisted contents of a tumour, but had a different pathological and clinical significance. With regard to the term "odontocele," he had in his hands the "Nomenclature of Diseases," published by the Royal College of Physicians, London, in 1896 and 1906. There were four classifications of cysts in 1896, but in 1906 there were only two classifications, and it seemed to him that, if a Committee of the Royal College of Physicians had been unable to agree about cysts, the whole question was in rather a chaotic condition. He introduced the term "odontocele" really to make things simpler.

Replying to Mr. Bennett's remarks, he said that if the X-rays had been used in the second case the diagnosis of malignant growth would never have been made. The swelling was diagnosed as a myeloid sarcoma and an operation was suggested, and that never would have happened if the X-rays had been used. With regard to the follicular odontome, he did not agree with Mr. Bennett, because a follicular odontome, as defined by Bland-Sutton, did not conform to the condition before them. As to the difficulties caused by the introduction of a term such as "Nasmyth's membrane," the words had passed into dental anatomy and pathology, and it was only in recent years that the membrane had become an important thing to consider. With regard to the degeneration of the stellate reticulum producing the amount of fluid found in such tumours, he quite agreed with him, because it seemed absurd to think that the stellate reticulum went on degenerating for twenty or thirty years, producing such a great quantity of fluid; but if a space was originally formed there might be an exudation of lymph from the neighbouring blood-vessels into the cavity owing to negative pressure, and he believed that was how the fluid went on accumulating in such cases. If he should have an opportunity of removing the maxillary canine he would do so.

Mr. James had used the word "dentigerous cyst" very largely in his criticisms; but Bland-Sutton had said that "it was a term which was used so very loosely that it should be discarded where there was any necessity for precision." Apparently a dentigerous cyst meant a cyst with a tooth in it, and therefore both the cases mentioned were dentigerous cysts from that point of view—i.e., from the standpoint of the *literal* meaning, but not from the view of the pathological or specified meaning. There was no doubt a lesion which had occurred in an early period, but what that lesion was, in both cases, it was difficult to find out. Mr. James seemed to think that the second case was a dental cyst associated with an unerupted canine. Of course it was not possible to know what pathological conditions existed round the canine before the appearance of the tumour, but apparently there had been no inflammatory condition of the periosteum of the unerupted tooth, and until that was proved it would be erroneous to call it a real dental cyst associated with an unerupted canine. The question arose, could petrification of the dental capsule occur in a few weeks' time? He thought not, and believed that the capsule which so closely covered the crown

of the tooth had existed for many years. He regarded the tooth follicle as rather an important structure amongst the lower animals, especially the Herbivora. The lives of the lower animals depended on their eating, and their eating on their teeth, and Nature had supplied them with a specially well-developed protection for the enamel and the dentine, and he believed himself the follicle was a protection to the tooth as it came through the gum. An odontocoele was a cystic tooth-tumour whose origin was different from that of either a dental or a dentigerous cyst, i.e., a follicular odontome.

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PATHOLOGICAL SECTION.

CONTENTS.

October 19, 1909.

	PAGE
The Present Position of the Neurone Doctrine in relation to Neuro-pathology (Presidential Address). By F. W. MOTT, F.R.S.	1
Rhabdomyoma of the Urinary Bladder. By S. G. SHATTOCK	31
A Note on Eosinophile Cells in the Exudate from Tick-bites on a Horse. By J. BURTON CLELAND	42

November 16, 1909.

Retroperitoneal Teratoma connected with the Spinal Canal. By RAYMOND JOHNSON and T. W. P. LAWRENCE	43
The Result of Double Oöphorectomy upon the Growth of the Uterus in the Rabbit. By S. G. SHATTOCK	51
Systematic Histological Examination of the Central Nervous System of a Case of Transverse Lesion of the Spinal Cord in the Lower Cervical Region. By SIDNEY V. SEWELL and H. HUME TURNBULL	61
A Method for Studying the Action of Blood-fluids and other Substances on the Leucocytes. By HELEN CHAMBERS	73

January 18, 1910.

An Experimental Investigation into the Origin and Cause of the "Pancreatic" (Cambridge) Reaction in the Urine. By P. J. CAMMIDGE and H. C. G. SEMON	79
By HERBERT FRENCH :—	
(1) Extensive yet Incomplete Fibro-caseous Disease of both Suprarenal Capsules in which Symptoms of Addison's Disease were not present	93
(2) Bronzed Diabetes, with Cirrhosis of both Liver and Pancreas; and Pigmentary Deposits in these Organs as well as in the Skin ...	94

	PAGE
A Chylous Cyst Excised from the Neck. By E. O. THURSTON ...	97
A Large Chylous Cyst of the Mesentery. By S. G. SHATTOCK ...	98
Action of Thyroid and Antithyroid Preparations. By WALTER EDMUNDS ...	100
The Influence of Oöphorectomy upon the Growth of the Pelvis. By S. G. SHATTOCK and C. G. SELIGMANN ...	102

February 15, 1910.

Attempts to produce Chondromatous or Osteomatous Growths by the Grafting of Fœtal Bones. By S. G. SHATTOCK, C. G. SELIGMANN and L. S. DUDGEON ...	127
Examination of the Brain of a Man who Lived Seven Hours after Receiving a Shock of 20,000 volts. By F. W. MOTT and EDGAR SCHUSTER ...	140

March 15, 1910.

Bile-producing Primary Malignant Tumour of the Liver ("Malignant Adenoma") : with Remarks on the Cells of Malignant Tumours retaining some of the Metabolic or other Functional Characteristics of the Cell-type from which they are derived. By F. PARKES WEBER ...	147
Amyloid Degeneration in an Adenoma of the Liver. By S. G. SHATTOCK ...	158
The Effect of the Injection of the various Tuberculins and of Tubercle Endotoxin on the Opsonizing action of the Serum of Healthy Rabbits. By R. TANNER HEWLETT ...	165
A Lipoclastic accelerating Action of Serum as an Index in Pathological Conditions. By J. A. SHAW-MACKENZIE and O. ROSENHEIM ...	168
Some Points in the Pathology of Acne. By ARTHUR WHITFIELD ...	172

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Pathological Section.

October 19, 1909.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

The Present Position of the Neurone Doctrine in relation to Neuro-pathology.

GENTLEMEN,—Allow me first to thank you for the great honour in electing me to the chair of this Section.

I was recently informed by the Senior Secretary that I should be expected to open this session by an address, and I propose therefore to give a general survey of the present position of the neurone concept, a subject in which I have been especially interested, as it is fundamental in neuro-pathology. During the last fifteen years it has given rise to much discussion, to many experiments and observations, and to new modes of improving our knowledge of the minute structure and development of the nervous system.

The neurone theory was at first accepted by most neurologists without hesitation, as it explained so many facts in pathology; but grave doubts were cast upon it, owing to observations and experiments made by those who disbelieved the interpretation put upon histological details by those who upheld the doctrine.

This controversy between scientific men seeking after truth was not without great advantages, for it led to the discovery of new methods of technique and new methods of embryological research, which now have finally established on a firm basis the *neurone concept*, which I may remind you was due to Forel, although reintroduced in the following terms by Waldeyer: A neurone is a nerve cell and all its processes,

including the protoplasmic processes or dendrons and the single-axis cylinder process with its cone of origin, its collaterals or side branches, and its terminal arborization. The *neurone theory* is that the nervous system consists of innumerable such anatomically independent nervous units in contiguity but not in continuity. There is interlacing of the processes but no network. The nervous units are genetically and trophically independent cellular organisms arranged in functionally correlated systems, communities, and constellations.

It is admitted by all that the nervous system consists of nerve fibres and nerve cells supported by neuroglia, and the main point of discussion

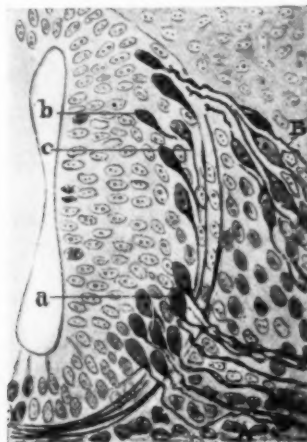


FIG. 1.

Section through the spinal cord of a chick embryo of three days. A, motor root; B, spinal ganglion; a, motor neuroblast; b, c, commissural neuroblasts. (After Ramon y Cajal.)

has been the origin of the nerve fibres. Waller more than fifty years ago showed experimentally that a nerve separated from its ganglion underwent degeneration, and every one has admitted there must be some intimate relation between the ganglion cells and the nerves. But it was asserted that when regeneration occurred it was owing to new fibres being formed by the cells of the sheath of Schwann. The question has long been in dispute whether regeneration takes place by a new outgrowth from the cell of origin or whether it is effected by the sheath cells. Likewise, there have been two opposing theories with

regard to the genesis of the nerve fibres, one being that the fibre is an outgrowth from the cell, the other that it is a product of the sheath cells. Consequently, the subject that I shall discuss first will be the genesis of the nerve fibre.

How is the nerve fibre formed? The answer given, first by Schwann and later by Kupfer, Balfour, Dohrn, and many others, and still more recently by Apathy, Bethe, and Schultze, is that the nerve fibre is the product of a chain of cells which extends from the nerve cells to the peripheral termination; they assert that these sheath cells secrete the fibrillæ within their protoplasm. Certainly such an interpretation of

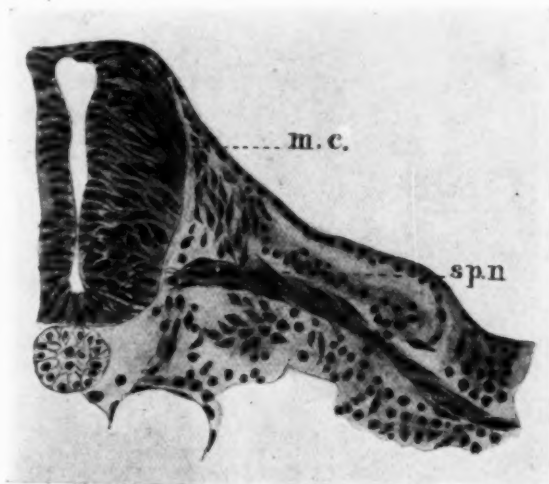


FIG. 2.

Cross section through a chick embryo of seventy-three hours, to show the beginning of a spinal nerve (*sp.n.*); *m.c.*, medullary cord. (After Bethe.)

the histological appearances could not be refuted till new methods of staining and new methods of experimentation were adopted to disprove this statement.

His, by his embryological researches, on the other hand, claimed that the axis cylinder and the other processes of the nerve cell were the outgrowths of the cell protoplasm. Then came the chrome-silver method of Golgi, which, in the hands of Ramon y Cajal, Kölliker, Retzius, Lenhossek, Van Gehuchten, and many others, apparently demonstrated

the fact that the whole nervous system consisted essentially of independent anatomical units, and the cylinder axon was an outgrowth of the nerve cell.

Ramon y Cajal was able to follow the development of the neurone from the neuroblast in all its stages, from the first appearance of the short nervous process up to the long outgrowth which formed the nerve

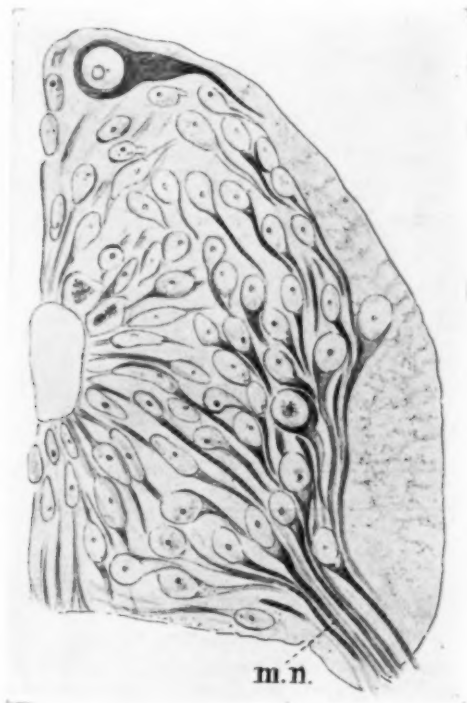


FIG. 3.

Cross section through the medullary cord of a salmon embryo, to show neuroblasts and motor nerve fibres (*m.n.*). (After His.)

fibre. His researches extended over the most varied motor and sensory regions of the central as well as of the sympathetic nervous system, and fully confirmed the researches of His. These results of the researches of Cajal were soon followed by important researches of Held, who was able to demonstrate that the nerve fibre is an outgrowth of the nerve

cell. Moreover, he accurately followed the origin and further development of the neurofibrils. They are situated, as already His, Bester, and Lenhossek had observed, in the interior of the neuroblastic protoplasm, first in the form of a loose skein, and then they grow out from the nerve-cell body into the nerve process. Held, however, like Hensen, finds that the nerve processes are not free, but grow into preformed tracts, which Hensen calls intracellular bridges, and he further believes that instances may arise where the neurofibrils of one cell grow into another cell, and, commingling with the fibres of that cell, pass out of

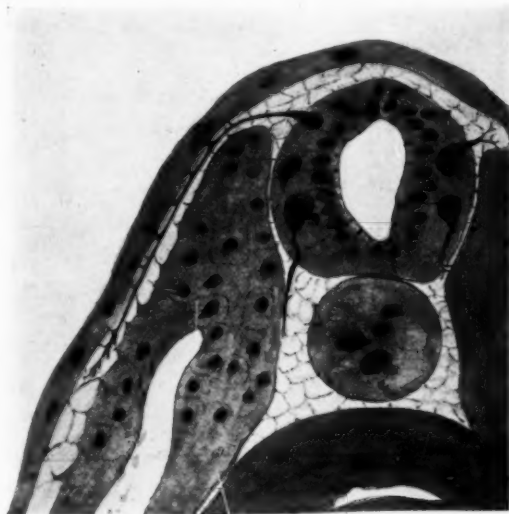


FIG. 4.

Semi-diagrammatic section through the spinal cord and adjacent organs of an axolotl embryo. The peripheral nerve fibre is seen arising from the ganglion cell; it does not grow out free into spaces between the cells, but into protoplasmic bridges or plasmodesmata which have been already formed by other cells. According to this theory the nerve paths arise through the transformation of these plasmodesmata into neurodesmata. (After Held.)

its axon process. As Verworn remarks, these facts are interesting histological details, but are completely indifferent for the neurone doctrine.

If we look at the figures (figs. 1, 2, 3, 4) given by the neuronists and the antineuronists, we can understand why this different interpretation

6 Mott: *Present Position of the Neurone Doctrine*

of the mode of development of fibres should occur. Ross Harrison truly remarks it is impossible to decide by microscopic examination of sections of embryos whether the spindle-shaped cells lying around the nerve fibres form the fibres, or whether they simply ensheath processes of the nerve cells. He states that his own observations upon the normal development of the salmon and frog had led him to a decided opinion in favour of the neurone conception, but the attitude of the opponents of the doctrine was such that the study of normal development would never prove convincing; consequently he set about finding some means of differential experiment.

Profiting by the knowledge of the transplantation experiments in tadpoles of Born, Ross Harrison planned a new and exact method of

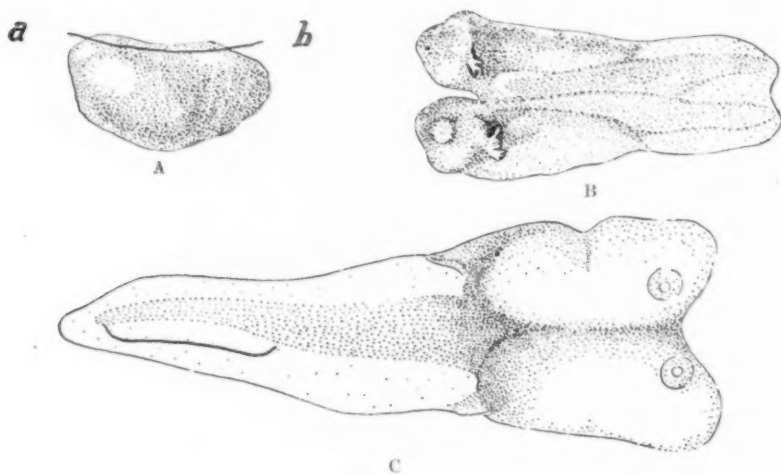


FIG. 5.

A, frog embryo 2.7 mm. long. The line *a-b* indicates the incision for the removal of the ganglion crest. B, C, two double embryos, from each of which the ganglion crest has been removed—B, two days after operation; C, six days after. (After Ross Harrison.)

study: namely, the removal in turn of each of the two structures in the developing embryo—frogs. Embryological studies had already shown that the source of the sheath cells was the ganglion crest; accordingly he removed this structure in the embryo *Rana esculenta* before any differentiation of nerve cells and fibres had occurred. The dorsal portion of the medullary tube and the ganglion crest was removed,

and the embryo was thus left with its nervous system as an open groove in its back, the walls of which contained the cells which would subsequently develop into the spinal motor cells. Two embryos so operated upon were joined back to back, so as to prevent regeneration processes vitiating the result (fig. 5). These animals were allowed to live a certain time and grow; they were then killed and examined carefully afterwards by serial sections. No spinal ganglia or sensory nerves were found, and

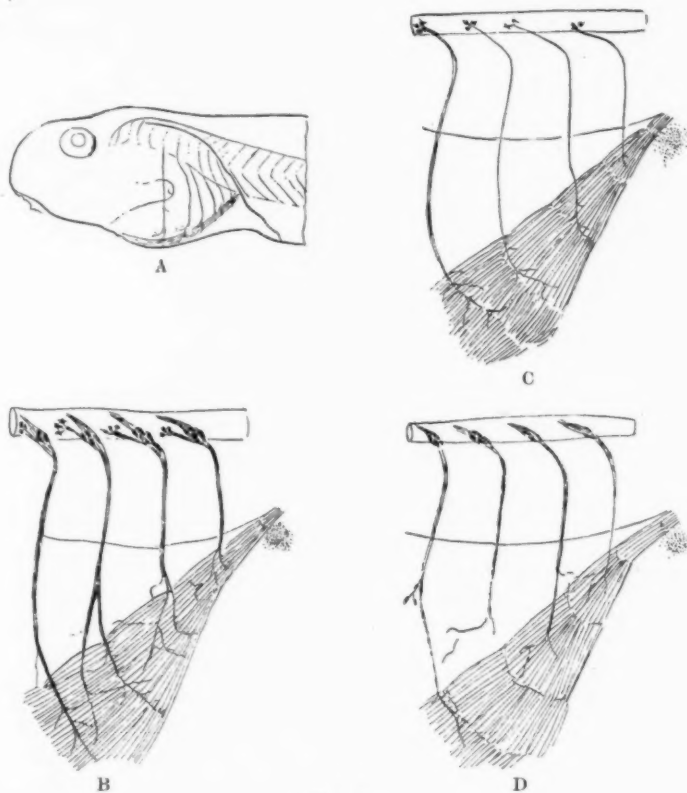


FIG. 6.

Diagrammatic views of the nerves in the abdominal walls of the tadpole. A, the body of a larva showing general arrangement of motor and sensory nerves, surrounded by sheath cells. B, arrangement in normal larva. C, arrangement in larva from which ganglion crest has been removed, only the motor nerves showing without sheath cells. D, arrangement in larva from which the ventral half of the spinal cord has been removed, showing only sensory nerves surrounded by sheath cells. (After Ross Harrison.)

what was of great interest was the fact that the motor nerves were found as fine protoplasmic threads extending from the spinal cord with not a single sheath cell around them (fig. 6, c). The nerve consisted simply of delicate fibrils. This experiment shows that the ganglion crest forms the posterior spinal ganglion, sensory nerves and the sheath cells. The conclusion is that the ganglion cells can form the nerve fibres without the aid of the sheath cells.

The next question Harrison sought to answer—and it is complementary to the last—was this: Can sheath cells without ganglion cells form the nerve fibres? He made the same incision as before, but

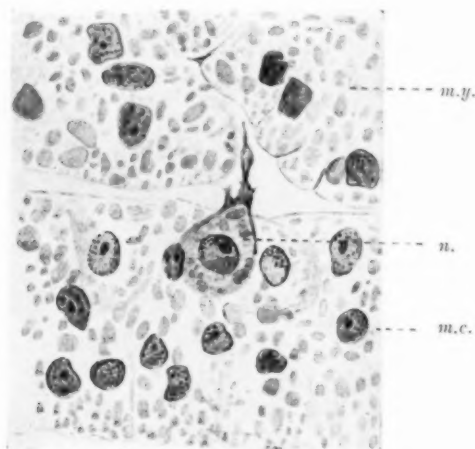


FIG. 7.

Portion of a horizontal longitudinal section through the spinal cord, *m.c.*, and portion of two muscle plates, *m.y.*, of a frog embryo. The cell, *n.*, with the branched process is a neuroblast, showing the first stage in the formation of the nerve fibre. (After Ross Harrison.)

instead of removing the ganglion crest he lifted it up and replaced it after removing with a fine pointed glass pipette the remainder of the medullary tube. The embryo developed normally, but remained almost motionless; subsequent examination showed that the sensory fibres and the sheath cells developed, but no motor fibres; although normally the motor and sensory fibres run together, and this would allow the fibres to the muscles ample opportunities of developing from the sheath cells if they were really the source of the motor fibres (fig. 6, d). He found,

moreover, that nerves separated from their cells of origin degenerate rapidly, and no signs of regeneration were observed.

The question may be asked, Does the ganglion-cell process extend as a nerve fibre to its peripheral termination, or is this process, as Hensen maintains, a differentiation of protoplasmic connexions already *in situ*? This view of Hensen, according to which protoplasmic bridges are supposed to be left between the dividing embryonic cells, has been supported by the difficulty of conceiving how it is possible for a nerve



FIG. 8.

Isolated cell from a piece of embryonic spinal cord growing in a drop of clotted lymph. The cell body, which is fitted with yoke granules, is sending out a hyaline protoplasmic process which undergoes amœboid movements. Drawn from a live specimen. (After Ross Harrison.)

fibre to grow out a long distance (and always to arrive at the right place) in the case of regeneration of the nerves of the foot after division of the sciatic nerve, for example. Harrison sought to solve the problem thus: He cut out the medullary groove that will form the spinal cord of an embryo before there was any trace of cell differentiation, and placed a small piece of it in lymph removed from the lymph-sac of a frog; the

preparation was put on a cover-glass, and mounted on a hollow slide and sealed with paraffin. The lymph clots almost immediately and holds the transplanted tissue in place. He has kept preparations alive five weeks and watched the development of the neuroblast, and he has seen the axon develop and grow (figs. 7, 8, 9). A somewhat similar structure to the cone of increase described by Cajal by his silver method of staining embryonic spinal cords (*see* fig. 10) can be seen. Probably it is also similar to the terminal bulb of regenerating axons found in the peripheral stump of a divided nerve. Harrison has observed a growth of $20\ \mu$ in

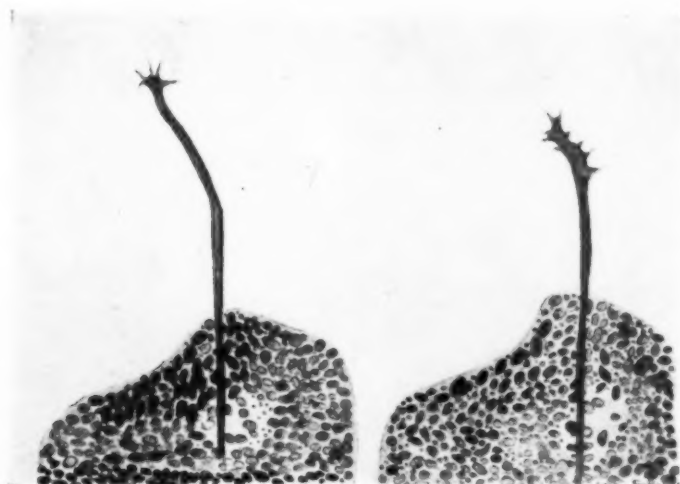


FIG. 9.

Two views, taken twenty-five minutes apart, of the same nerve fibre growing from a group of embryonic spinal-cord cells into the lymph. (After Ross Harrison.)

twenty-five minutes, as shown in the figures. Control experiments with other tissues of the embryo gave no such results. These remarkable observations of Ross Harrison show beyond question that the nerve fibre begins as an outflow of hyaline protoplasm from cells situated within the central nervous system. This protoplasm is actively amoeboid; retaining its pseudopodia at its distal end, the protoplasm is drawn out into a thread which becomes the axis cylinder of a nerve fibre. Although Harrison has disproved the theory that the sheath cells

form the essential portion of the nerve—namely, the axis cylinder—yet he fully admits, as all those who uphold the neurone doctrine do, that the cells of the sheath of Schwann play an important rôle in the nutrition and production of the nerve fibre. These experiments of Harrison place the outgrowth theory of His on the safest of all foundations—namely, that of direct observation. It is, moreover, interesting and important to note briefly that Marinesco, Goldstein, Minea, and Nageotte have observed in transplanted spinal and sympathetic ganglia of warm-blooded and cold-blooded animals an outgrowth of fibrils from the ganglion cells. As Verworn truly says, the embryological basis of the neurone doctrine has been established beyond dispute. The formation

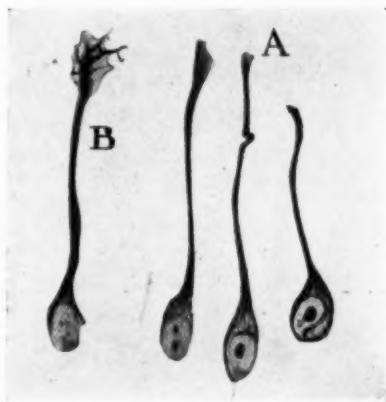


FIG. 10.

A, neuroblasts stained with silver nitrate. B, neuroblast impregnated by the Golgi method. Growth cone appears well marked at the top of B. (After Ramon y Cajal.)

of connexions of one neurone with another by fibrils, the passage of fibrils from one nerve cell through another nerve cell, are matters of detail which in no way shake the fundamental principle of the neurone concept—namely, the cell and its processes form a nerve unit which has a genetic independence. According to Kölliker, all the nerve fibres arise from nerve cells of the central nervous system and the ganglia. The peripheral nerve fibres are surrounded by special cells, constituting in the adult the sheath of Schwann. The latter appear when the axis cylinder is developed, and constitute for it a superficial envelope. These

sheath cells all arise from the mesoderm and multiply by karyokinesis. According to Froriep, however, the cells of the sheath of Schwann of the peripheral nerves are probably, like the neuroglia cells, of ectodermal origin. Certainly Harrison's experiments seem to support Froriep's views.

The anatomical observations of Cajal and others I have named regarding the genetic independence of the neurone have thus been fully confirmed by the researches of Harrison. The next question is, Are the sheath cells of no further use than to enclose the axial core; in fact, is there a complete trophic independence of the nerve cell and its processes? Experimental and clinico-anatomical observations indicate that the sheath cells play an important part in regeneration after injury of the axon. To prove this statement we have only to consider the



FIG. 11.

Wallerian degeneration of regenerated nerve fibres in a cat. Marchi method of staining. (600 diameters.)

difference in the results on the extra-medullary and intra-medullary course of the axons of the posterior spinal ganglia after section of the posterior roots. Sherrington and many other observers have described fine medullated regenerated fibres around the cord—that is, central to the seat of injury—but only isolated observations have shown a few regenerated fibres in the exogenous systems of the posterior columns.¹ We may correlate these facts with the presence of sheath cells in the

¹ Again, although Marinesco, Minea, and Fickler have seen attempts at formation of regenerating fibres after transverse lesions of the spinal cord, there is no restoration of function, because there is no re-establishment of connexion with the appropriate neurones.

extra-medullary posterior roots and the absence of the same in their intra-medullary course. It was shown by Gudden that evulsion of a cranial nerve caused atrophy and disappearance of the nuclear cells of origin, and this method in his hands and that of his pupils, von Monakow and Forel, laid the foundation of much of our knowledge of the nuclear origin of the cranial nerves. Simple section of the nerves, as Nissl, Marinesco, Van Gehuchten, and others have shown, produces chromolytic changes, but as a general rule the cells do not undergo atrophy. It may be assumed that evulsion was a more serious injury

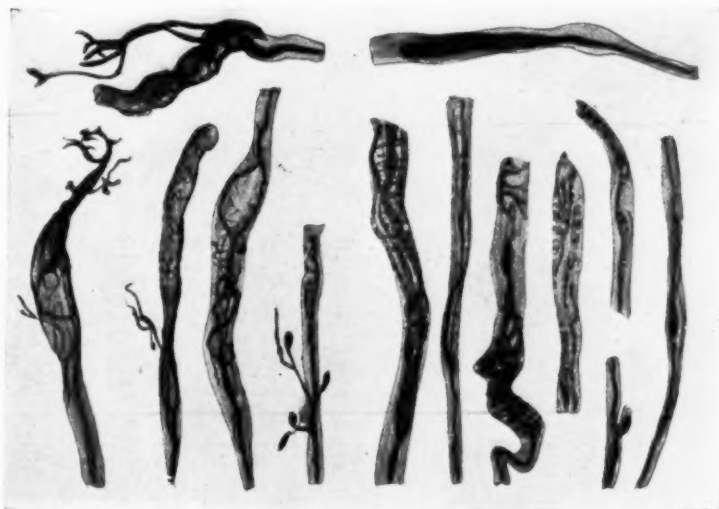


FIG. 12.

Regenerating nerve fibres from the end of the central nerve stump of the sciatic nerve of a dog, taken from six to forty-eight hours after cutting the nerve. (After Perroncito.)

and destroyed not only all the sheath cells of the neuraxon up to the point of origin of the nerve from the central nervous system, but was also a much more serious injury of the neuraxon itself.

Before proceeding to describe the recent advances in our knowledge concerning the changes which take place in the central and peripheral ends of a divided nerve, it will be well first to consider the changes in the nerve cells of origin as revealed by the Nissl method.

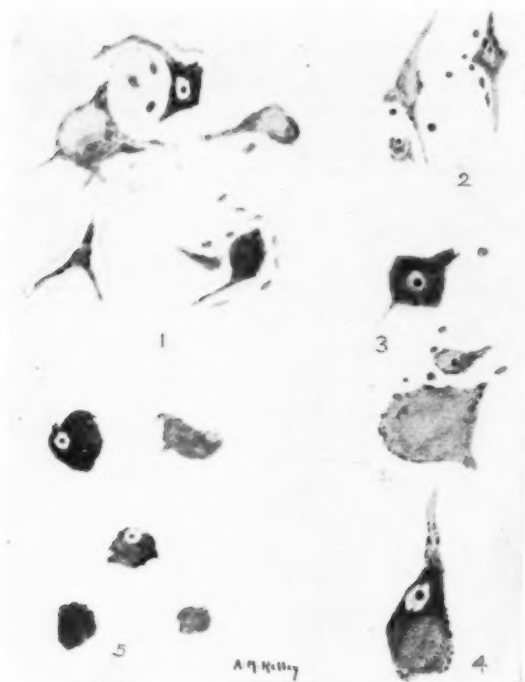
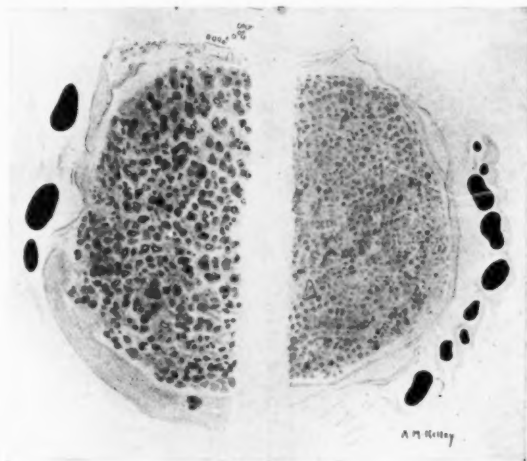


FIG. 13.

(1) Group of anterior horn cells from a monkey's spinal cord on the side in which section of the sciatic nerve had been performed. On the opposite side hemisection and section of all the lumbo-sacral roots had been performed, in addition to section of the sciatic nerve. Yet there were as many altered spinal motor neurones on the one side as the other, thus showing that the cause of this alteration was the section of the nerves, and that interference with the passage of stimulus had had little to do with the trophic condition of the cells. It will be observed that some cells have the normal Nissl pattern. (2) Cells of the cortex cerebri in phosphorus poisoning of the rabbit. It will be observed that one cell shows considerably more chromolytic change than the other. (3) Cells of the anterior horn of the cervical enlargement in a case of peripheral neuritis caused by chronic lead-poisoning. The perinuclear chromatolysis and eccentric condition of the nucleus is very evident, and the appearance presented is the same as by the large cells in fig. 1. (4) A large Betz cell of the cerebral cortex with eccentric nucleus and chromatolysis specially marked around the axon cone of origin; from the same case of lead neuritis. (5) Five cells showing various degrees of eccentricity of the nucleus and chromatolysis of the posterior spinal ganglion cells from a case of alcoholic neuritis.

CHANGES IN THE CELLS DUE TO LESIONS OF CRANIAL AND PERIPHERAL NERVES.

Nissl first published (1892-4) detailed accounts of the changes in the nerve cell (observed by his method of staining) produced by section of the nerves. Twenty-four hours after section of the facial nerve in the rabbit there is a reaction in the cells of the facial nucleus. The chromatic substances commence to disappear in limited regions of the cell body, and after two days, instead of presenting the blocks of stainable substance, now known as the Nissl granules, there was observed



(a) FIG. 14. (b)

(a) Regenerating motor bundle of a cat's sciatic nerve ninety-one days after the nerve was divided and sutured. Marchi's method of staining. The transverse section shows well-marked myelination in the majority of the fibres; it was taken from the upper end of the nerve. (Magnified 220 diameters.) (b) The same bundle 3 in. lower down. Myelination much less advanced. (Magnification 220 diameters.)

a fine dust of coloured particles. By the third day the process had extended to the dendrons. At the end of five or six days the cell has changed its form, it has become rounder, the prolongations less visible, and the protoplasm looks as if sprinkled with dust like particles of stainable substance. During this time the nucleus has changed its position and become eccentric. Nissl also studied the alterations in the cells

of the posterior spinal ganglia produced by section. Marinesco repeated and confirmed Nissl's experiments, using the hypoglossal nerve. He was followed by Ballet and Dutil, Lugaro, Van Gehuchten, Flemming, and many others. The changes observed may be thus summarized: Swelling of the cell body with slight increase of volume of the nucleus and of the nucleolus; the cell becomes rounder and the prolongations in their turn swell; there is disintegration of the Nissl granules, a process which begins in the central region of the cell near the axon-cone of origin. The first changes are observed twenty-four hours after section, and they



FIG. 15.

Olive-shaped swellings at the end of nerve fibres, growing downwards from the central end twenty-one days after the nerve had been divided. Coiled-up fibrils can be seen in these swellings. (After Marinesco.)

progress until the whole perinuclear region exhibits this chromatolysis and the Nissl granules on the protoplasmic processes disappear. The nucleus becomes eccentric in proportion to the amount of chromatolysis. These changes are due to alterations of the osmotic conditions of the cell caused by the injury to its axon process, whereby fluid is absorbed from the ambient medium. Marinesco names this characteristic change

"perinuclear chromatolysis"; a similar appearance of the nerve cells is seen in neuritis. I have observed it in all cases of alcoholic polyneuritis and in lead neuritis (*vide* fig. 13). This reaction of injury to the nerve cell when its axis-cylinder process is injured is in direct confirmation of the correctness of the neurone doctrine, for if the sheath cells formed the axon it is much less likely that the nerve cell would show such changes, which may even proceed to their atrophy and disappearance. It is remarkable that when a nerve is divided only some motor spinal ganglion cells are so severely damaged that they do not recover but undergo atrophy and disappear, so it is found in polyneuritis some cells hardly show any change, while others are so injured as to be incapable of recovery, and eventually atrophy and disappear (fig. 13). Again, in transverse lesions of the spinal cord, as shown by Pusateri, Marinesco, and Gordon Holmes, the Betz cells of the cortex undergo similar chromolytic changes which may proceed to atrophy, but the cells are affected very unequally, for we may observe two Betz cells lying side by side, the one seriously damaged and the other hardly affected at all. Drs. Sewell and Turnbull have investigated a case of transverse lesion of the spinal cord due to fracture of the spine at the level of the fifth cervical. The boy died at Charing Cross Hospital eight weeks after the injury. The Betz cells at the top of the ascending frontal convolution showed all degrees of chromatolysis, from cells with hardly perceptible changes to complete disappearance of the Nissl granules. I have observed also in experimental poisoning by botulin, absinthe, phosphorus, and other poisons, also in acute and chronic poisoning of human beings, that the neurones are not equally affected. Still more striking is the fact that, after experimental ligature of all four arteries, two carotids and two vertebrals, or two carotids, one vertebral, and one subclavian, whereby an experimental cortical anæmia in lower animals—for example, monkey, cat, and dog—was produced, the cells of the same type—for example, the Betz cells—were not equally affected; relatively only a few were so damaged as to undergo destruction. The conclusion is that the specific vital energy of some cells is greater than that of others, just the same as the individuals of a society are not all possessed of the same vital resistance to injury or poison; but as we know that the vital resistance of the individuals of a society is largely a matter of nutrition, fatigue, and powers of conservation of energy, so probably it is with the neurones. Those in which nutritional equilibrium is, either from inherent defect, fatigue, or other causes, in a low state will suffer most.

I have also observed this chromatolysis of the Betz cells very marked in cases of chronic alcoholic and chronic lead polyneuritis; it was more

marked to my mind than could be accounted for by the small amount of degeneration of the pyramidal tracts, and I cannot help thinking that it was the effect of the poison on the whole system of efferent neurones concerned with reflex and voluntary movements plus, the parenchymatous and peripheral interstitial polyneuritis (fig. 13). All the neurones, however, do not undergo destructive atrophy; probably only those in which the nucleus is extruded are incapable of recovery. My experience has taught me that even in severe cases of polyneuritis recovery may result from continued passive movements of all the joints and massage of the muscles. The avoidance of pressure and weight of the bed-clothes in rendering a foot-drop permanent, the instruction of the patient to make voluntary effort by aid of attention and vision, and try to assist the operator making the passive movements, the placing the feet against a board while lying in bed, and the daily placing the feet on the ground in the effort to feel the weight of the body supported by the limbs, all tend, by favouring incoming and outgoing stimuli, to open up the old paths or to restore function by new paths. This is no mere hypothesis, for it is based upon the fact that Munk found that monkeys in which the motor area had been removed much more completely regained movements when placed in a cage large enough to enable them to spring about and take free exercise.

Upon this assumption, therefore, that stimulus exercised an important influence upon regeneration of nerves, Halliburton and I carried out a series of investigations to see if regeneration of the motor nerves was interfered with by cutting off, so far as we were able, stimuli from the spinal motor neurones of a limb. The following procedure was adopted: unilateral section of the posterior roots from the third cervical to the third dorsal inclusive was performed, the animals used being monkeys. There was as a result a marked hypotonus of the limb, which hung useless as a flail, and the animal did not use it for refined voluntary purposive movements. Either the median or the ulnar nerves on the two sides were then divided and sutured. There was apparently no difference in the time taken for regeneration on the two sides, nor could I discover any more marked changes in the anterior horn cells of one side than of the other. Again, the motor area of the right side of the brain was removed in an animal in which those same posterior roots had been divided; the nerves were divided on both sides, and the result was the same as in the previous related experiment. A third experiment was performed; a unilateral section of the lumbo-sacral roots was combined with hemisection of the spinal cord in the mid-dorsal region

and both sciatics were subsequently divided and sutured. Even in this experiment we could not claim that all sources of stimulus to the spinal motor neurones of the side of the double lesion had been removed. Still, the greater part of the paths exercising any influence upon the discharge from the spinal motor neurones had been removed, and if stimulus really did play an important part in the nutritional activity necessary for re-

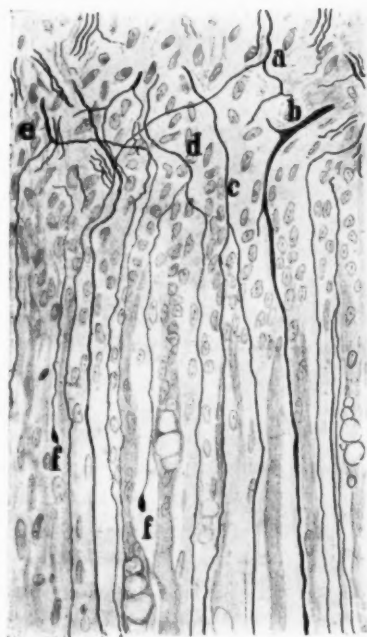


FIG. 16.

Regenerating nerve fibres of a cut nerve. The young fibres *a*, *b*, *c*, *d*, *e*, growing from the central end, can be seen passing through the cicatrix down into the peripheral portion between the proliferated sheath cells. Some of these are seen to bifurcate—for example *d*, *c*, *e*. One branch of *d* terminates in an olive-shaped bulb, *f*, the other proceeding onwards. It is possible that the former is arrested in its progress, and coils up to form the olive. (After Ramon y Cajal.)

generation of the motor nerves, after section of the axons of the motor neurones, it might be certainly expected that regeneration would be delayed on that side; but it was not, and this accords with the fact that

the characteristic changes in the ganglion cells were not more apparent on the side of the double lesion than on the opposite side. A careful examination of sections of the spinal cord failed to show more ganglion cells presenting chromolytic changes on one side than the other. In order to avoid any personal bias, I tried to see if I could ascertain on which side the double lesion had been made by an examination of the anterior horns; but I failed entirely, although when I shifted the specimen to the posterior cornua it was quite evident from the atrophy of the fibres and changes in the cells. Fig. 13 shows two degenerated cells on the side opposite to the spinal and root lesions.

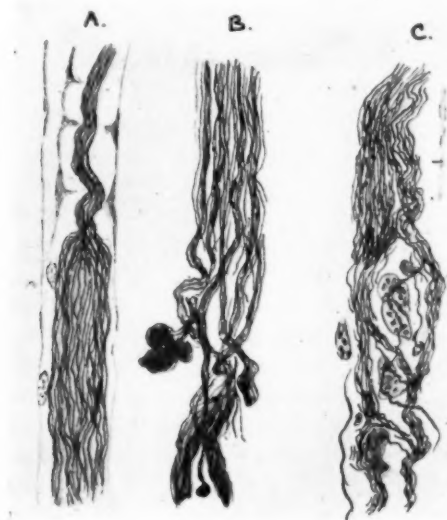


FIG. 17.

Section of the sciatic nerve of a dog five and a half days. Details of degeneration in the central end. A, axis cylinder with a nearly normal appearance in its upper part, whilst in its lower part it is considerably swollen, and the neurofibrils are very apparent, and like a tress of hair. In reality it is a reticulated structure made evident by an increase of the interfibrillary and perifibrillary substance. B, axis cylinder swollen and with the neurofibrils dissociated at the upper part, and reunited in diverging cords towards the upper third. Some of these end in a terminal bulb. C, the axis cylinder swollen in a still more marked manner, and consisting above of tresses of neurofibrils. These reunite into cords below, which surround more or less the debris of a degenerated axis cylinder. (After Marinesco.)

The trophic activity of the spinal motor neurones is therefore, in all probability, independent of stimulus arriving from other neurones. They possess, indeed, a trophic autonomy. The observations of Harrison support this idea; moreover, this accords with the neurone doctrine of genetic and trophic independence. Direct injury of any part of the neurone will cause changes but not injury of neurones, with which it is functionally, but not nutritionally, correlated.

The axon consists of a number of conductile fibrils, and the more of these fibrils which are severed the greater will be the injury to the



FIG. 18.

Hypertrophied fibres of the central cut end of a nerve with very obvious fibrils presenting an abundant lateral and terminal division. Some of the collateral and terminal ramifications are seen ending in a small or large bulb; others have a free extremity. The proliferated sheath cells are very obvious. (After Marinesco.)

neurone. It is possible that fibrils constituting an axon may not come entirely from one cell in every case; there may be, as Held suggests, in some instances a commingling of fibrils proceeding from different cells to form the axon of a nerve; in such a case it is conceivable that the

reaction of injury would be less than when all the fibrils of a cell are divided.

We have briefly discussed what happens to the cell when that portion of the neurone which is termed the axon is injured; I will now consider the changes which occur (*a*) in the peripheral portion of the nerve, and (*b*) in the central end of a divided nerve, in the light of modern researches, especially as revealed by the new silver method of Cajal, which shows that a neurone consists of fibrils which pass from the dendrons through the body of the cell to the axon, and consequently may be assumed to act as the conductile element of the neurone.

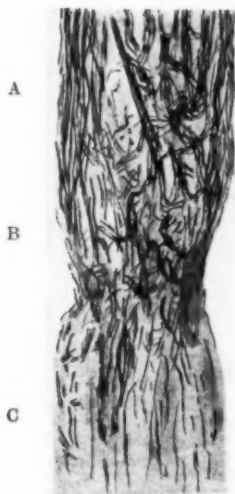


FIG. 19.

Longitudinal section of the sciatic nerve of a new-born dog eight days after section. A, central end. B, cicatrix. C, peripheral end. (After Marinesco.)

The appearances presented by fibres undergoing degeneration when stained by the Marchi method are well known. I may call your attention to fig. 11, which is of considerable interest for another reason to which I will briefly refer. The following experiment was performed by Halliburton and myself: A small piece was resected from a nerve in which regeneration had taken place, and histologically the nerve was shown to possess many fine myelinated fibres. Ten days later the animal was killed, and the portion of the nerve below the resection was

examined; no myelinated fibres were found, but a number showing Wallerian degeneration. If the sheath cells had been mainly instrumental in the formation of the new regenerated fibres, resection of the piece of nerve should have had no influence upon the nerve which had developed from the sheath cells.

The changes which occur in the central end of the cut nerve were very incompletely known before Cajal introduced his silver method, and the histological observations of Perroncito, Cajal, and Marinesco by this method have thrown a new light upon the process of regeneration. Perroncito, three hours after section of the nerve, has found in the central end of the nerve very fine collateral ramifications of the axon, terminating, after a short course, in very delicate filaments. After eighteen hours he has observed in the central end most of the anatomical conditions indicative of regeneration, which, previous to his researches, had been looked upon as late productions of the axis cylinder—namely, the plaque, the ring, the button, and the helicoidal formation (fig. 12). These observations of Perroncito show that active nutritive changes commence almost directly after the injury before the sheath cells divide. Marinesco has confirmed the observations of Perroncito, and has found that within twenty-four hours of section of the sciatic nerve very important and interesting phenomena of regeneration occur. These phenomena are the results of an increased nutritive activity associated with a swelling and hypertrophy of the large axis cylinders, whereby the constituent nerve fibrils which make up the axons become evident. The network or skein of fibrils are clearly seen, owing to a great increase of the interfibrillary substance, and a longitudinal dissociation of the bundle of fibrils takes place. These skeins of fibrils may run side by side or intercross one another. The various appearances of the fibrils resulting from this longitudinal dissociation are represented in fig. 17, and are very different from one another. Between the tangled skeins of dissociated fibrils there may be offshoots of more delicate fibrils forming plexuses. Some of these delicate fibrils, as fig. 18 shows, terminate in a little olive or cone of growth. This process of longitudinal fibrillary dissociation of the axon leads to the formation of a number of embryonic fibres, from which develop a number of young axis cylinders. These young fibres can be followed through the central cut end to the cicatrix. Some are seen to terminate in a button, olive, or club-shaped extremity, forming a cone of growth which penetrates between the more or less longitudinally-arranged sheath cells (fig. 16). At the termination of the cut ends there is a cellular exudation and new-formed blood-vessels; this

undergoes organization, and at the end of six or seven days forms a bridge which unites the two cut ends for the passage of the young regenerating fibres. But in proportion as the axon fibres penetrate the cicatrix, they diminish in numbers and their branches are less numerous. The new fibres may be seen to diverge, intercross, and form a felt work, as seen in fig. 19. A certain number of them, however, can be seen taking a straight course and descending vertically in the peripheral stump. Cajal considers that the terminal bulbs correspond to the cone of growth of the developing axon in the embryo or pseudopodia in the advancing axon of the neuron observed by Harrison in his experiments. Probably Cajal is correct in maintaining that the terminals of the new-formed young fibres correspond to an amœboid phase of very active growth, which assures the neurotization of the peripheral end of the divided nerve. After the stage of active ramification or dendro-amœboidism numbers of terminal clubs of the young fibres are seen. These traverse paths rendered free to their passage between the sheath cells (fig. 16).¹ Marinesco thinks that these terminal clubs only indicate a delay or retardation of chemiotropism of the sheath cells. The shape of these terminal bulbs, which are so striking a feature of the regenerating nerve fibrils, is variable; in structure they consist of a network or twisted skein of fibrils in the form of an olive or bulb and an interfibrillary amorphous substance (fig. 15). It is difficult to decide whether these bulbous terminals represent cones of growth or a twisting up of a fibril on account of an obstacle to the growth or nutrition of the nerve fibril.

If sections be made of the junction of the central and peripheral ends at successive periods of time, three stages can be seen: (1) The sprouting of large numbers of new fibres from the central cut axon; (2) the penetration of the bridge of young vascular connective tissue joining the cut ends by the young nerve fibres; and (3) the penetration and growth downwards of these young fibres between the proliferated sheath cells of the peripheral portion (fig. 16). There can be no doubt, then, that the new nerve fibres depend upon an outgrowth from the central cut end of the neuraxon.

What part, then, we may ask, do the sheath cells play in regeneration?

To answer this question it is well to consider some of the appearances presented by the peripheral fibres which are undergoing Wallerian degeneration when stained by the Cajal silver method. The lesions are the same in all animals, but the moment of their appearance depends upon the species, age of the animal, and severity of the injury, which

¹ Professor Marinesco has kindly forwarded me the sections from which the figures were drawn.

has caused the solution of continuity of the nerve. The changes appear first at the upper end and are propagated peripheral-wards through the whole extent of the nerve below the lesion. The degeneration does not affect all the fibres equally. The first change is a swelling of the axis cylinder whereby the fibrillary network is displayed. The neurofibrils are visible and appear thickened in their course. But very soon the axon appears only as a dark-stained finely granular cord (fig. 20, A). In place of neurofibrils there is a dark mass of fine granules. Marinesco terms this condition "axolysis." He believes that the fragmentation of

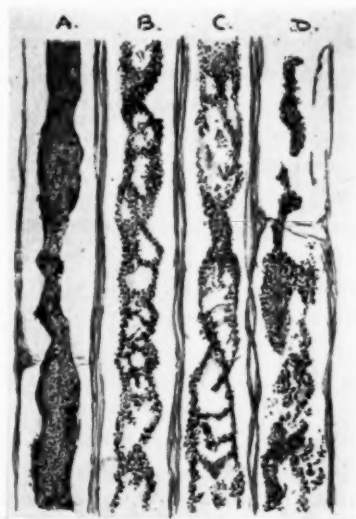


FIG. 20.

Represents four fibres showing different degrees of axolysis. A, the axis cylinder no longer shows any neuro-fibrils, but consists of abundant fine granules disseminated in the interfibrillary substance. B, formation of cavities and vacuoles in the interior of the axis cylinder, in consequence of resorption of the granules. C, the same condition more advanced. D, fragmentation and breaking up into small pieces of the axis cylinder. (After Marinesco.)

the myelin sheath and axis cylinder constitute connected phenomena dependent upon the same cause—namely, the formation of a digestive ferment on the inner surface of the sheath of Schwann. He moreover considers the ferments formed are analogous to the proteolytic and lipolytic ferments of the pancreatic juice, for Bokay has found that the pancreatic juice splits lecithin up into cholin, glycerophosphoric acid

and fatty acid. It is the oleic acid which stains black with the Marchi reaction. Resorption occurs in this softened and swollen axon, causing vacuolation. The vacuolation becomes more marked as the process of resorption continues, and this results in fragmentation of the axon, and finally the fragmented axon is completely destroyed and absorbed (fig. 20, B, C, D). The sheath cells undergo rapid proliferation and exercise a phagocytic action upon the products of degeneration, while others arrange themselves in a longitudinal direction to receive and attract the rapidly growing young fibres which have come from the central cut end. The experiments of Forssman appear to show that the products of degeneration have a chemiotropic influence upon the developing fibrils of the cut central end of the nerve.

Forssman cut the tibial nerve and brought the central cut end into relation with the divided peroneal and tibial nerves, which he placed in a celloidin tube; two months later he found the same number of regenerated fibres in each of the nerves. This may support the view that the sheath fibres exercise a chemiotactic influence, but Forssman made another series of experiments showing that this chemiotropism depends upon chemical attraction of the degenerated myelin. His experiments were conducted thus: He divided the central end of a nerve and placed each half into two separate celloidin tubes; one was filled with emulsion of liver and the other with brain emulsion; the fibres all grew in a direction towards the tube containing the brain emulsion. It may be concluded from this experiment that the degenerated myelin of the peripheral portion of the nerve attracts the young fibres and the sheath cells ensheath them.

Forssman also showed that the fibres of the central end do not take the line of least resistance, but the direction to which chemiotropism attracts them. Cajal, however, asserts that it is the attraction of the protoplasmic strands formed by the sheath cells which are arranged in dense fusiform bundles, and Cajal and Marinesco believe that these neurilemmal cells secrete a chemiotropic substance capable of exciting amoeboid movement in the pseudopodia of the young regenerating axons, and, having attracted, direct and nourish them. Marinesco claims to have proved this by the following experiments. He took a piece of nerve in which the sheath cells are dead; it was obtained either from a dead animal or by resection of a piece of nerve from a living animal, which was allowed to remain a sufficient time in serum for death of the sheath cells to have taken place. This was connected to the central cut end of the sciatic nerve of an animal. To the central

end of the other divided sciatic nerve he connected a piece of nerve kept alive in Locke's oxygenated serum. In the former case, when the sheath cells are dead there is no chimiotropic influence upon the fibres of the central cut end, whereas in the latter, if the resected nerve is from the same species, the fibres of the central end grow in. As the cell processes grow down into the new sheath an inter-action takes place between the new axis cylinder and its sheath, leading to the formation of intervening myelin. Moreover, Halliburton and I found, in regenerating nerves, a much larger number of myelinated fibres and the myelin sheath of the fibres thicker near the point of section than in a remote portion of the nerve (fig. 14, A, B). This was an indirect support to the outgrowth theory, and has also been observed by Langley and Anderson.

It may be assumed, therefore, from what I have said, that the trophic and genetic independence of the neurone has been established. A question still remains, and that is, How are these nervous units connected with one another; if they are independent, how are they functionally correlated? This has, from the foundation of the neurone concept, been a point in dispute. Although many valuable researches have been published, still we are far from understanding the problem. The Golgi method appeared to show that the neurones are arranged in systems and communities in contiguity but not continuity, and numbers of experiments and observations were made to show that the dendrites exercised amœboid functions, and little buds or gemmules on these processes of the cells were supposed to appear and disappear owing to the contractility of the protoplasm, thus leading to varying degrees of contact influence of one neurone upon another. But there is no proof that these gemmules exist in the living cells, although the amœboidism of the branching processes of the neurones does seem possible as an explanation, when Harrison's experiments combined with Perroncito's observations are considered. But, as Verworn justly remarks, we must be careful not to generalize upon insufficient data, for the physiological connexions in the nervous system of the lowest types of animals as compared with the higher vertebrates are different; take, for example, the simple reflex processes in the lower invertebrates and the complicated processes of association in the human brain. In the invertebrates there can be no question that Apáthy and Bethe are right in asserting that there is a fibrillary continuum through several ganglion cells.

But does a continuity exist in the vertebrates? In my Croonian Lectures on the Degeneration of the Neurone, I referred in the following

words to some important researches of Held which had been put forward to disprove the neurone theory. "In my opinion, however, one can still accept the neurone theory and admit the truth of Held's observations—namely, that the terminal arborization of the axis-cylinder process of one neurone forms protoplasmic concrescences by fusion with the cell body and dendrons of another. This, of course, implies continuity of the protoplasm of one neurone with another, but trophically and genetically the two are independent, and it is merely a question of degree of contact of the protoplasm of one with the other. Held agrees with other investigators that in embryonic tissues, and even in early life, the neurones are entirely independent of one another. This independence he can determine by a line of demarcation at the points of contact due to a difference in refraction. This refractile limiting line is, however, not demonstrable in the adult, and he comes to the conclusion that during the process of growth the protoplasm of related neurones fuses." Verworn states that the pictures which Held publishes are not clear enough to satisfy him. On the other hand, the possibility is undeniable that where the axon of one neurone is in contact with the dendrites of another there is a "receptive substance," which Sherrington has termed the synapse; this substance may possess certain physiological properties which have a similarity with those of the end organ in striped muscle—for example, easy fatiguability, great summation, capacity for excitations, and specific reactions for certain poisons, &c. "But it remains undecided whether the acceptance of such a synapse having the above related physiological properties is necessary for the explanation of the physiological processes. In any case great difficulties exist both for the acceptance of the synapse as for the acceptance of a continuous path of conduction. It is a fact that conduction of the stimulus through the reflex arcs only occurs in the physiological, and not in the opposite, direction. Yet it can be demonstrated that the nerve possesses conductivity in both directions; therefore the necessary conditions for this difference of conductivity must be in the centre. Where the conditions are situated and how they are affected, the future must decide." It is admitted that the lower we descend in the zoological scale the more obvious is the fibrillary continuum, and the simpler and less varied are the motor adaptations to environment. We may assume, therefore, that in the upward development of the animal series with the complexity and refinement of motor adaptation there coexists a neuronic independence. We may ask the question, What would be the advantage in the evolution of the nervous system of neuronic independence? In

the attempt to answer such a question it is desirable to look at it from a broad biological standpoint. What are the properties of nerve tissue, and how does it differ from other cell protoplasm? We may say it is irritable and conductile, but we know that the protoplasm of the carnivorous plants—the sundews and Venus flytrap—is irritable, conductile, contractile, and secretory, and these plants are capable of exhibiting reflex action without a nervous system when the appropriate stimulus is applied. This reflex action, however, differs from that of the plant-like animals, with their diffuse network of neuro-muscular cells, in speed of reaction.

Consider, moreover, the advantage of speed of voluntary action initiated in the brain by the impulse having to pass through the conductile fibrils of only two nervous cells in the production of motor reactions of the arm, leg, face, and tongue. But if there existed a fibrillary continuum from the cerebral neurone through the anterior spinal neurone, how could the anterior motor spinal neurones form the "final common path" for a multitude of different muscular activities?

The idea that the neuraxon of a Betz psycho-motor cell is connected by its conductile fibrils with one anterior spinal motor neurone is a textbook myth, which, instead of making the nature of voluntary movements to the thoughtful student clear, only makes him ask the question, Are the number of fibres in the pyramids of the medulla equal to the sum of all the fibres in the anterior roots? Clearly the fibrils, then, of the Betz cells must be connected with a number of spinal motor neurones forming the final common path for a multitude of different voluntary muscular activities. Moreover, we know that there is not a direct connexion between the upper cerebral neurones and the lower spinal motor neurones, for intercalary neurones with axons which do not leave the grey matter intervene, and the terminal arborization of sensory afferent neurones, cerebral, mesencephalic, and bulbar efferent neurones, combine with spinal association neurones through the intercalary neurones in augmenting and inhibiting the outflow of energy of the spinal motor neurones, whereby the most varied, refined, and complex muscular reactions in adaptation to environment are effected. The nervous energy is expended in the grey matter of the brain and spinal cord.

There are many other interesting points concerning the neurone which I might, had time permitted, have spoken about—for example, the significance of the nodes of Ranvier and the perifibrillary protoplasm; the theories in respect to the functions of the interfibrillary chromo-

philous substance of the ganglion cell and the dendrons. Is this chromophilous substance the source of nerve energy, and, if so, is it used at the seat of its formation, the nerve cell? or is this substance, which in the dead cell corresponds to the Nissl granules, a store of energy-producing substance in the latent state analogous to the proferment of secretory glands—a substance which is transformed into the active energy-producing substance at the terminal arborizations of the neurone? I must, however, rest content if the facts and arguments which I have related have satisfied you that the neurone doctrine, pronounced dead only a few years ago, now rests on a surer basis than ever, this satisfactory result being due especially to the introduction of new methods of investigation by Ramon y Cajal and Harrison.

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Rhabdomyoma of the Urinary Bladder.

By S. G. SHATTOCK.

Vesicae urinariae tumor e telâ musculari transverse striatâ constructus (Rhabdomyoma).

SUMMARIUM.

E dysuriâ atque urinae retentione aegrotabat puer, haematuria semel modo observatâ: ex uraemiâ mortuus est.

Vesicâ post mortem incisâ tumores polyposi e membranâ mucosâ orti intus apparuerunt.

Hos e telâ musculari transverse striatâ constare scrutatio probat microscopica.

Inveniuntur fibrae musculares e cellulis ingentibus multinucleatis gradatim evolutae, cellularum nucleis in cytoplasmatis margine dispositis.

Neoplasmata nullas malignitatis notas in structurâ penitiori monstrant; nec invaditur tunica visceris muscularis.

Telae muscularis striatae evolutionem in hoc situ quomodo licet intelligere?

In plerisque certe heteroplasia heterotopiam significat, i.e., cellularum dislocationem abnormem de structuris quibusdam in telas adjacentes.

In vesicae urinariae exemplo cellulae embryonales, ut opinor, sphincteris vesicae externi in telam vesicae submucosam vagatae sunt.

Hujus musculi ad normam includuntur fibrae quaedam in glandulae prostatae substantiâ apud vesicae cervicem.

Illam thesem sustinent hae considerationes: (1) Tumores ad vesicae partem inferiorem limitantur; (2) Ingeniti probabiliter insuper erant.

TUMOURS of striped muscle fibre are, wherever they may occur, of extreme rarity, and the growth of such a new formation in so unexpected a position as the urinary bladder will fully justify the record of the following specimen.

The bladder, which is that of a child aged 2 years, was presented to the Museum of the Royal College of Surgeons by Mr. C. S. Wallace, who

has kindly allowed me to record it. For about its lower two-thirds the interior is raised and deeply lobulated by the growth of a series of closely-applied sessile tumours, smoothly covered with mucous membrane. A portion of the new formation projects into and occupies the vesical orifice of the urethra, but the mucosa of the prostatic portion itself is free of disease. The right ureter is considerably dilated, its aperture being completely surrounded by the growth; the wall of the left ureter is thickened, although the canal is not obviously enlarged. The muscular wall of the bladder is somewhat hypertrophied. The prostate is normally developed.

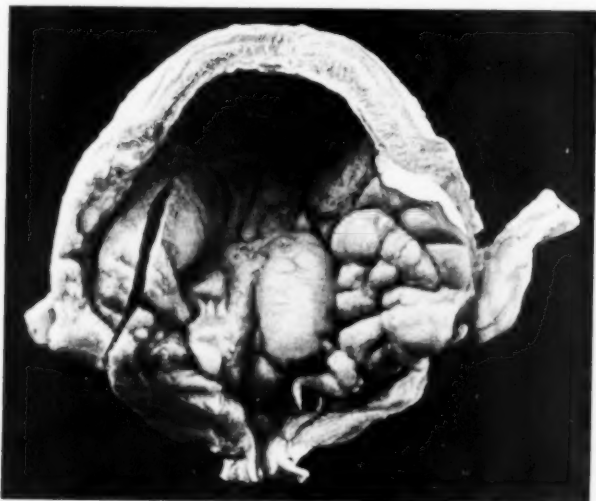


FIG. 1.

Pueri (annorum duorum) vesica urinaria, pariete posteriori diviso. Monstrantur tumores in cavum projecti qui e telâ musculari transverse striatâ præcipue constant. Magnitudinis naturalis.

The specimen referred to in the text, the bladder being laid open, and viewed, from behind. For its lower two-thirds the interior is raised and deeply lobulated by the growth of a series of closely applied sessile tumours, which consist chiefly of striated muscle. (Museum of the Royal College of Surgeons. Natural size.)

The child (A. B.) was admitted into the Children's Hospital, Shadwell, under the care of Mr. C. S. Wallace, on November 22, 1906, with a history of four months' pain on micturition; on one occasion, three

months before admission, the passage of blood had been noticed. There was no family history of importance.

When admitted the patient was well nourished. There was no pain on palpating the abdomen, and nothing was detected by it; rectal examination was equally negative. On the day following admission an attack of retention occurred; the urine was drawn off and the bladder sounded, but nothing was discovered to explain the retention. This was the only occasion on which retention took place. Micturition gave, apparently, no pain. The urine was neutral; specific gravity, 1015; it contained a thick cloud of albumin and a fair number of pus cells, but no blood or casts.

A fortnight later diarrhoea and vomiting set in; these resisted all treatment, and rapid wasting ensued. Unconsciousness supervened and all the symptoms of uræmia, death occurring in a state of coma, but without convulsions. The amount of urine passed daily was from 15 to 25 fluid oz. No hæmaturia was observed.

HISTOLOGY.

The tissue as studied in vertical sections of one of the chief of the tumours consists throughout of a close felt work of extremely delicate and wavy fibrils, well provided with normal connective-tissue cells. The fibrillæ are not disposed in bundles, but form, by their interlacing, a uniform felt, the whole structure being that typical of a soft fibroma. The cells are as devoid of any arrangement as the fibre, and, as they are subject to no compression, show large oval, as distinguished from fusiform, nuclei. The free surface of the growth is raised in low, simple elevations, without being papillary. At the free or vesical surface there is a comparatively narrow zone in which the cells are more thickly clustered, and amongst them have wandered a certain number of polymorphonuclear leucocytes, the appearances being indicative of an inflammatory process involving the surface of the growth, a result attributable to the cystitis present during life. Well-developed arterioles are distributed in the new tissue. There is nowhere any suggestion of a sarcomatous transformation being in progress, the more cellular tissue being limited, as already stated, to the free surface immediately beneath the epithelium.

None of the proper muscular wall of the bladder appears in the section, and as no mucosa remains differentiated from the new growth, the latter, it may be concluded, has arisen in the mucous and submucous

coats. So far the growth would be a soft fibroma. It may be added that there is no neurofibromatosis.

But here arises the real matter of histological interest: the fibrous tissue is throughout, but in different degrees, pervaded by striated muscle fibres in all stages of development. In some spots these form a slender, wide-meshed net, in others they are sparse; and fields may be selected where there are none. They are least abundant in the deepest part of the growth, and they are most densely aggregated in the region of the free surface, where they are disposed in closely intersecting fasciculi. The striated fibre varies somewhat in the details of its structure.

As studied with one-sixth objective, the closely intersecting bundles last referred to consist of long, narrow fibres of no greater breadth than the cells of unstriated muscle. The cross-striation, however, is quite unequivocal, for whilst the longitudinal may be hardly or not recognizable, the other is sharp, parallel, and extends regularly across the whole breadth of the fibre. The great majority of the fibres present it. But besides such there is elsewhere striated fibre in the ordinary stages of development, and in positions where the latter can be clearly followed by reason of the fibres being isolated in the midst of the delicate connective tissue. These appear as multinuclear masses of protoplasm of diverse form according to the direction of the section, the nuclei lying at the periphery and being very irregularly distributed with regard to the length of the developing fibre; in some areas the sarcoblasts occur in groups, in others singly. The middle or widest portion of such developing fibres is of considerable thickness.

Where divided transversely the cytoplasm appears finely granular, and in such views the nuclei are invariably disposed peripherally in it, sometimes on one side in a horseshoe form. When viewed longitudinally these multinuclear elements are irregularly fusiform, and the cytoplasm is, in a certain number, doubly striated—i.e., both longitudinally and transversely.

By the elongation of such structures and the separation of their nuclei, there result the long, doubly striated fibres which are present in other parts, and many of which present a series of nuclei parted longitudinally by considerable intervals. These fibres may be of striking length, and it is noteworthy that the nuclei of such are not centrally but peripherally disposed; nor have the latter the length of those of unstriated cells. This peripheral disposition of their nuclei alone would serve to differentiate such fibres from the unstriated kind, although in

actual breadth they may not exceed that of well-developed plain cells. In one cross-striated fibre the sarcolemma was brought into view by the rupture and retraction of the included substance.

Such slender fibres at times run in twos and threes in a wavy course, but remain discrete, with narrow and not always regular interspaces. None of the fully developed striated fibres attain the breadth of those of common voluntary muscle.



FIG. 2.

Structurae e sectione microscopica tumorum unius selectae. Monstrantur fibrae quaedam musculares, transverse striatae, in gradibus diversis ad normam evolutae.

From a microscopic section of one of the tumours, showing a selection of developing and developed striated muscle fibres. The smallest element is a cross-section of a multinucleated sarcoblast with peripherally disposed nuclei. The supporting basis of fine connective tissue is not shown. (One-sixth objective.)

In the deeper part of the growth a few isolated bundles of unstriped muscle cells occur in the fibrous tissue, but these seem to be distinct in nature from the rest of the muscular tissue, and may be regarded, I think, as stray elements of the muscularis mucosæ, or deeper muscle, which have become entangled in the new formation.

It is well known that the cells of unstriped muscle may present a longitudinal striation and indistinct traces of a (spurious) transverse one; and it is necessary, in order to anticipate criticism, to state that a study of the microscopic sections under $\frac{1}{2}$ oil immersion fully confirms the foregoing account, and makes it clear that there is a large amount of striated fibre present in the growth. The transverse darker markings appear as broad equidistant bands of perfect regularity, and in some the intervening lighter zone is crossed by a finely punctated line (Dobie's line). The broader darker zones in some cases are resolvable into bundles of sarcoous elements. Many of the long multinucleated fibres, however, whilst they exhibit good longitudinal fibrillation, show no transverse striation; and this is true even of some of the broad multinucleated fusiform elements. Even in the course of the same developing, elongated, and sometimes varicose fibre, the cross marking may be well pronounced in certain places, whilst the longitudinal is alone discernible in others. And lastly, even the most slender fibres, such as those which are gathered in intersecting groups, show in cross-section a difference from unstriped cells in that when a nucleus happens to be divided it is not centrally located, but lies at the periphery of the cytoplasm, which is, moreover, particularly granular from the presence of the fibrils included within it.

Let me next refer to a second specimen (No. 3,693) which is also in the Museum of the Royal College of Surgeons, and which is even a purer example of rhabdomyoma than the foregoing. Here, too, the growth takes the form of multiple polypi, and comes from a child who was likewise aged about 2 years. The lower part of the viscus is thickly beset with polypoid growths, the smaller of which arise from the neck and project into the prostatic portion of the urethra; most of these are cleft into secondary processes, or subdivided, without being, however, papillary. Higher up the growths are of considerably larger size but fewer in number, and are attached by broader bases, and merge into sessile thickenings of the mucosa. The upper half is quite free. At the apex of the bladder, on the external aspect of the muscular coat and covered by the peritoneum, there is a sessile nodule about as large

as a haricot, in which there is an irregular central cavity; the inner surface of the organ is here free of new growth. The muscular wall of the bladder is hypertrophied. The ureters are dilated. The child was aged $1\frac{1}{2}$ years when first under observation; for more than a month previous to this, micturition had been frequent and painful, and was attended with great straining. No calculus was detected by the sound. A perineal cystotomy was eventually carried out, and portions of the growths were cut away with scissors as they appeared in the wound, into which they were forced by the straining efforts of the patient. Death occurred forty-eight hours later. The specimen is engraved in a "Treatise on Calculus," by Mr. J. G. Crosse.

The histological study of these polypi, in sections made vertically to and including the free surface, shows that they consist almost entirely of well-developed, though slender, transversely striated muscle fibre. In the deeper part of the growth the fibres lie in a basis of soft fibrous tissue with few pertaining cells; but at and for some way below the free surface they constitute the whole of the structure, the intervening finely fibrillar connective tissue being quite insignificant in amount and provided with but few proper cells. All the fibres show the cross-striation. There are no spindle or other cells suggestive of a sarcomatous formation. Many of the muscle fibres show a very pronounced longitudinal fibrillation in addition to the cross-marking. The component fibrils are particularly clear in the transverse sections of the fibres. In addition to this there occur amidst the fibres short, broader, fusiform masses of protoplasm enclosing groups of nuclei, and representing developing striated fibre; in these the stages of striation are traceable just as in human foetal muscle. Generally speaking, the muscle fibres are not thicker than unstriped cells, though a certain number exceed such.

Mr. J. H. Targett¹ has referred to this specimen, but the presence of multinucleated developing muscle fibre has escaped his notice, although he remarks that in some of the "long spindle cells" a transverse striation can be recognized, but that the microscopical features are not so distinct as could be wished. This reserve I cannot explain, unless it is to be attributed to the use of a faulty objective and the omission to study the sections with an oil-immersion.

The small nodule at the apex of the bladder, mentioned in the description, is at first suggestive of a metastatic growth; yet microscopically it shows no striated fibre, and nothing at all resembling the

¹ *Trans. Path. Soc. Lond.*, 1896, xlvii, p. 291.

tumours within the bladder. The cavity in its centre is surrounded with fibrous and fibromuscular tissue, and is lined with a cellular exudate. It lies in the situation of the urachus, with which structure it is probably related; and I should be inclined to disconnect it altogether from the other growths in the sense of its being a metastasis. So abundant is the striated muscle, and so small the fibrous tissue in the intravesical growths in this case, that the new formations must be classed without qualification as rhabdomyomata.

There is a third specimen in the Museum of the Royal College of Surgeons which, as interpreted in the light of the two foregoing, must be placed in the same category. The preparation (No. 3,692), which is one of Hunter's, is a lateral section of the bladder and urethra of a young girl. Numerous lobulated polypi arise from the mucous membrane and nearly fill the cavity. The largest has a base nearly 2 in. in diameter; the others have narrow pedicles; two of them have grown from the neck of the bladder into the urethra, and have protruded beyond its external orifice, their ends being ulcerated and flocculent. In places the growth is low and sessile, as around the orifice of the right ureter. The muscular wall is uninvaded and readily separable from the diseased. The upper half of the viscus is unaffected. The preparation is figured in Baillie's "Illustrations," fasciculus vii, pl. 4, fig. 2. A study of microscopic sections, under $\frac{1}{12}$ immersion, discloses a considerable number of multinuclear giant-cells, with granular cytoplasm, the nuclei in all cases being peripherally disposed. In some, when viewed in the longer axis, the cell body shows a well-marked longitudinal fibrillation. The chief bulk of the tissue consists of long, slender, intersecting fibres in not very well-defined bundles, the intervening tissue being very scanty and of a delicate, open, fibrillar kind. The giant-cells first referred to lie amongst the fibres, sometimes congregated in clusters. The fibres in question, though not in general broader than unstriped cells, differ from such in the fact that they are provided with more than a single nucleus. Oftentimes a series of nuclei, in longitudinal apposition, lie along the fibre; in these fibres, moreover, there is a well-marked longitudinal fibrillation. Some of the fibres presenting these characters are wider than the rest, and some are swollen out in parts of their length, there being a cluster of nuclei in the swollen parts. Notwithstanding these characters, which show that the fibres belong to the cross-striated kind, the transverse striation is present in but few; where it is to be seen, however, it is quite distinct, the component

fibrillæ, which are traceable individually, being regularly subdivided into darker and lighter segments.

The presence of multinucleated cells in this specimen was noticed by Mr. Targett (*loc. cit.*), but he did not attempt to give any interpretation to them.

And, to close the list, there is a fourth specimen in the Museum of St. Thomas's Hospital (No. 2,163). This is the bladder of a young male child. Over its lower half the mucous membrane is raised in low, smoothly rounded ridges, from which gradations may be traced to the prominent polypi which are clustered about the neck of the bladder. Some of these hang from long, narrow stalks, and appear to have been forced into the urethra beyond, which is much dilated. The mucous membrane of the prostatic portion of the urethra is beset with similar but more minute outgrowths. The microscopic sections of one of these polypi show that it consists of delicate connective tissue, which is traversed by numbers of long, narrow fibres, furnished with eccentric or peripheral nuclei. Some of the fibres are of striking length, and a considerable number exhibit a typical transverse striation; the nuclei are disposed at intervals in the course of the fibre. The fibres produce a wide-meshed net and run in intersecting groups composed of a few elements only, and these not closely compacted but loosely accompanying one another. Those that present no transverse striation are of the same kind in that they are of marked length and furnished with multiple nuclei. Fusiform, multinuclear sarcoblasts occur abundantly with the larger fibres, conformably with which they are disposed; some of these show an exquisite cross-striation. The tissue in which the fibres lie is that of a soft fibroma, and is composed of a loose felt work of very delicate, wavy fibrillæ, furnished with a moderate number of connective-tissue cells. Histological marks of malignancy are entirely absent.

REMARKS.

Such an anomalous specimen naturally raises the question, Where can the striated fibre in a submucous vesical tumour come from? Speaking generally, it is simpler to explain the presence of heteroplastic structures as due to a process of dislocation having taken place during development, from some adjacent organ or part in which such structures normally occur, than to regard them as produced *ab initio* at the site in which they are found. Of dislocation and the shifting of parts during

normal development embryology teems with examples. But as for new growths, it is usual, e.g., to view the striated muscle fibre found in rhabdomyosarcomata of the kidney in infants, as having been originally derived from the embryonic muscle-plates with which the metanephros is so closely related; or, if the tumour does not involve but only lies against the kidney, to regard the tissue as having been misplaced into the precursor of the permanent kidney, the Wolffian body. The correctness of such a view is strongly supported, if it is not proved, by the straying of adrenal tissue (apart from tumour formation) witnessed in the kidney, or in the liver, the broad ligament, and elsewhere. Analogous displacements are well-recognized phenomena in the development of the spinal cord, where the grey matter may be "heterotopic," or abnormally disposed in the white; and the same term might be usefully extended to other examples of the same phenomenon. Heteroplasia in tumours is in most cases heterotopia.

In connexion with the bladder, then, it may be remembered that striated fibre is to be found normally in the foetus, the child, and also in the adult, around, and to some considerable depth in, the substance of the anterior or superior portion of the prostate, the structure in question constituting the sphincter vesicae externus of Henle. As Henle's figures show,¹ the fibres completely encircle the apex of the prostate, but in the cross-sections carried further back they appear only on the anterior or upper aspect. From the front of the gland they pass obliquely upwards and backwards round the neck of the bladder in the groove between the viscus and vesicula seminalis, constituting a superficial or external, though not strongly marked, sphincter. The absence of the striated fibre from the lower or posterior aspect of the prostate in transverse sections carried through the more proximal part of the gland, is due to the obliquity of the muscle. Some excellent recent figures showing the external vesical sphincter of Henle, as it appears in transverse sections of the prostate, are given by Mr. C. S. Wallace in his work on "Prostatic Enlargement."² Some of the fibres (as shown also in Henle's figures) lie well within the prostatic substance.

The explanation which I venture to put forward in the case of rhabdomyomata of the bladder is that the growth of muscle fibre has arisen from vagrant sarco blasts which have abnormally extended or

¹ "Handbuch der systematischen Anatomie," Braunschweig, 1866, ii, p. 380; and "Grundriss der Anatomie," Braunschweig, 1889, Tafel cl.

² *Oxford Medical Publications*, 1907, figs. 5, 8, 9, 14, 17.

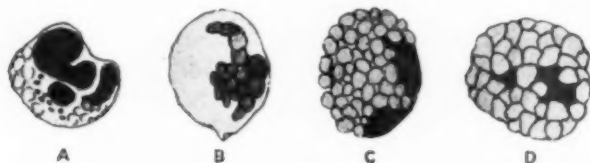
have been displaced, beyond their usual deeper limits, into the sub-epithelial tissue of the bladder. Such a misplacement or ingress might occur the more readily during the earlier stages of development in the male since the growth of the prostatic glandular tissue from the urethra would either not as yet have taken place or would offer but little obstacle as a barrier to it.

There are two considerations which may be advanced in favour of this hypothesis. One of these is that in three of the four cases the tumours were met with in infants, and were probably congenital. The child in Mr. Wallace's case was only two years old, and there was a history of four months' pain on micturition. In Crosse's case the patient was aged $1\frac{1}{2}$ years when first under observation, and for more than a month micturition had been painful and frequent. And the bladder in St. Thomas's Hospital Museum is that of quite a young child. In all of these the tumours were almost certainly present at the time of birth, but had not then attained sufficient size to produce noticeable symptoms. Hunter's case was that of a young girl, but the size and number of the growths shows that the disease must have been many years in progress, for the neoplasm is not malignant. And, secondly, it is significant that in all four cases the disease is limited to the lower portion of the bladder; the upper half of the viscus is, in every one, quite free of growth. That this is equally so in Hunter's case a recent examination of the specimen shows: although the whole of the vesical cavity is filled, no tumours are attached to, or arise from, any part of the upper half of the organ.

A Note on Eosinophile Cells in the Exudate from Tick-bites on a Horse.

By J. BURTON CLELAND.

A NUMBER of camels were recently imported from India into the north of West Australia. Attached to some were large ticks kindly identified by Dr. Nuttall as *Amblyomma egyptium*. These rapidly spread temporarily through the country, frequently attaching themselves to horses and cattle. Seeking on these especially the tender parts, such as around the sheath and between the legs in horses, the ticks produced much irritation with the exudation of a slimy, bloody discharge. Films made from this discharge showed the presence of numerous red cells and leucocytes. The majority of the latter were eosinophile cells, the others being polymorphonuclear leucocytes (with the lobes of the nuclei each containing a dark central spot), lymphocytes, and large mononuclear cells.



Eosinophile cells from exudate from tick-bites in a horse. **A B**, cells containing one granule in each; **C D**, normal eosinophile cells.

The large number of eosinophile cells, with the large round granules so characteristic in the horse, is of interest. The number of the granules in the cells varied from 1 to 30, perhaps occasionally more. Some consecutive numbers were 25, 28, 30, 26, 23, 13, 3, 16. In looking through the fields, two cells were found containing only one granule in each. Does not this variation in number from one upwards support the suggestion that these granules are in fact a secretion, and that the cells containing very few are those in which the secretion has been almost completely discharged or in which the new formation of granules is just commencing after the exhaustion of the cell? An alternative supposition is that the granules, when few, are the result of phagocytosis from injured eosinophile cells.

Pathological Section.

November 16, 1909.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

Retroperitoneal Teratoma connected with the Spinal Canal.

By RAYMOND JOHNSON and T. W. P. LAWRENCE.

THE boy from whom the specimen to be described was obtained was exhibited, when aged $2\frac{3}{4}$, before the Society for the Study of Disease in Children on November 16, 1906. In the preceding August a swelling was first noticed in the left loin, and a few weeks later the child was admitted into the Victoria Hospital for Children. The lumbar spine presented a well-marked lateral curvature with its concavity directed to the left (fig. 1). The left half of the abdomen was occupied by a large elastic tumour which passed back into the loin, and, as far as its relations were concerned, might well have been considered as a tumour of the kidney (fig. 2). There were also two small fluctuating prominences in the left loin external to the border of the erector spinæ. Although the nature of the case was evidently obscure, it was regarded as probably one of tuberculous disease affecting the left side of the bodies of the lumbar vertebræ and thus causing a lateral, rather than the ordinary angular, curvature. According to this view, the swellings in the loin and abdomen were thought to be chronic abscesses. An exploratory incision was, accordingly, made over one of the smaller swellings in the loin, which proved to be one of a number of cysts extending amongst the muscles of the back and containing sebaceous matter. It was thus clear that this part of the tumour had the characters of a dermoid.

At a later date the abdominal part of the tumour was explored by an oblique incision in the loin. It was at once evident that a complete removal was impossible, and the operation was completed by opening

a large projecting cyst and inserting a drainage-tube. After the operation the inner wall of the cyst prolapsed considerably through the opening, and, becoming inflamed, produced an appearance strikingly like that due to prolapse of the mucous membrane through a colotomy opening.

The child became progressively weaker and died on April 14, 1907. There were, throughout, no evidences of paralysis of the lower limbs.

The specimen consists of the adjacent parts of the dorsal and lumbar portions of the spinal column and the lower three ribs of the left side,

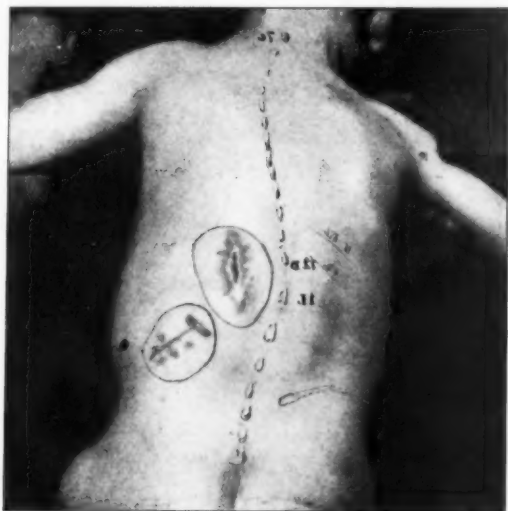


FIG. 1.

Showing the lateral curvature and the position of the prominences formed by the tumour posteriorly.

together with a large teratoma which is connected with the left side of the spinal column. The part of the spinal column preserved presents a very pronounced lateral curve, the concavity of which, directed to the left, is occupied by the inner border of the tumour. The tumour reaches upwards to the lower border of the tenth rib, and the eleventh and twelfth ribs lie on its posterior surface. It measures 10 cm. vertically, 10 cm. transversely, and 7.5 cm. in an antero-posterior

direction. Viewed from the front (fig. 3) the outer two-thirds at least of the tumour are seen to be formed by a large, partially-collapsed cyst. From the front of this a circular portion of the thick, fibrous cyst-wall has been removed and a vertical section made through it. In this way, after the removal of a considerable amount of sebaceous matter containing short, pale hairs, there is exposed a large, solid projection into the interior of the cyst. The section through this solid projection shows it to have a very varying structure in different parts; dense fibrous bands, small bluish areas like cartilage, and cyst-like spaces, containing an almost gelatinous material, being recognizable. Between the large cyst above described and the spinal column, and lying in the concavity

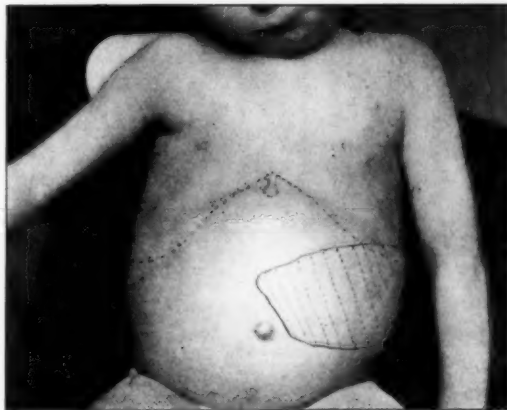


FIG. 2.

Showing the anterior limits of the tumour.

of the latter, are three rounded projections of the tumour. A slice has been cut from the front of the lowest of these, showing it to consist partly of cystic, partly of solid portions. Embedded in the solid portion is a hard, conical structure having a general resemblance to a tooth, but, on examination, proving to be a curved plate of bone enclosing a central mass of cartilage.

Viewed from behind (fig. 4), the specimen shows the large cyst, before mentioned, projecting beneath the extremities of the last two ribs. In this situation the cyst was incised at the time of the operation. Its wall has become everted and its inner surface appears as a smooth,

folded membrane, somewhat resembling prolapsed bowel. At one spot a small polypoid outgrowth springs from the everted cyst-wall, and superficial ulceration has occurred in places. The space between the everted portion of the cyst and the spines of the vertebræ is occupied by several smaller projections of the tumour. Most of these have thin, semi-translucent walls; in one, however, the walls are thick and skin-like, and a few brown hairs spring from its inner surface.

The laminæ of the right side have been removed from all the vertebræ, thus displaying a rounded projection of the tumour into the spinal canal. This projection, which measures 5.5 cm. in vertical

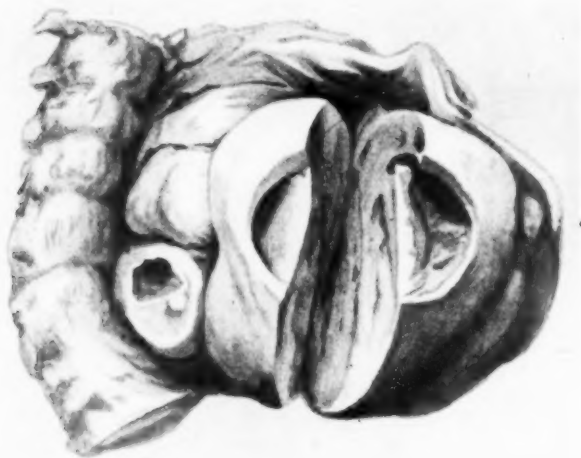


FIG. 3.

The tumour seen from the front.

extent, has a thin translucent wall and is filled with sebaceous material. The theca spinalis is displaced to the right and is closely applied, but not adherent, to the projection, which presents a deep groove in this situation. The spinal cord terminates at the usual level, and it and the nerves of the cauda equina, though pressed against the wall of the spinal canal, are normal to the naked eye, having evidently been protected from pressure effects owing to the soft consistence of the projecting mass and to the groove above mentioned. The spinal canal in the situation of the

projection presents a slight fusiform enlargement. Embedded in the dense fibrous wall of the lower and posterior part of the tumour is an elongated mass of bone of irregular form, measuring $2\frac{1}{4}$ cm. in length. This is attached by fibrous tissue to the transverse process of the third lumbar vertebræ, and its outer extremity is tapering and terminates in a thin, recurved, and pointed process of cartilage. Those parts of the tumour-which are in contact with the vertebræ are closely adherent to the outer surface of the bones.

It is not possible without further dissection to ascertain precisely what are the defects in the spinal column which have permitted of the



FIG. 4.

The tumour seen from behind.

protrusion of the tumour into the spinal canal, or whether the protrusion is through the intervertebral foramina. The bodies of the vertebræ and the laminae of both sides can be seen to be normal, and any defect that may be present would appear to have its seat in the pedicles of one or other of the last dorsal or first lumbar vertebra or of both of those bones.

The left kidney is moulded to the anterior surface of the tumour, having a convex anterior and deeply concave posterior surface. On

section its structure appears to be unaltered. The retroperitoneal tissue above the kidney is fibrotic and the suprarenal body cannot be recognized. The anterior surface of the tumour was readily separable from the retroperitoneal tissue except at one spot just above the kidney, where it was adherent over an area about 1 cm. in diameter.

MICROSCOPIC EXAMINATION.

The lining of the cyst is composed of a layer of stratified epithelium varying in thickness in different parts. Malpighian layer, prickly cells,



FIG. 5.

A microscopic section from the solid portion of the tumour, showing a central lumen with columnar epithelium; outside this a layer of fibrous tissue, containing numerous narrow bundles of smooth muscle fibres; outside this again thick bundles of smooth muscle fibres arranged circularly.

stratum granulosum, and stratum corneum are recognizable, though one or more of these may be absent in different places. Papillae are ill-developed or absent in the sections examined. The tissue subjacent to the epithelial layer differs in different parts: in some places it consists of tracts of fibrous tissue alternating with layers of fatty tissue and areas having a myxomatous character; in other parts it is composed of a soft, loose areolar tissue, from which fat is absent. In places, groups

of coiled tubes are present, surrounded by a network of capillaries, evidently sudoriferous glands. No sebaceous glands or hair-follicles are present in the sections examined, though it is obvious they must be present in other parts of the cyst-wall.

A section of a more solid portion of the tumour removed from the loin at the time of the operation shows a structure which probably represents intestine. This structure presents a central lumen of complicated form and somewhat resembling that of the Fallopian tube in transverse section (fig. 5). The space is bounded by a layer of cylindrical epithelium resting upon a stratum of fibrous tissue in which are



FIG. 6.

A microscopic section from the solid portion of the tumour, showing a central lumen with columnar ciliated epithelium; outside this a fibrous layer, containing numerous mucous glands; outside this again three bars of cartilage—one above the lumen, the others, of which only the extremities are visible, at the sides.

scattered bands of smooth muscle fibres. Outside the fibro-muscular layer, and in places separated from it by a layer of vascular areolar tissue, is a broad encircling layer consisting of bundles of smooth muscle fibres separated by narrow tracts of fibrous tissue. In general the bands of muscle fibres in this layer are arranged circularly around the central lumen; some, however, have an irregular disposition.

A section from the solid body contained in the main cyst shows a structure which probably represents some part of the respiratory tract (fig. 6). In the centre is an irregularly shaped slit-like lumen bounded by an epithelial covering composed of several layers of cells, the innermost of which is cylindrical and ciliated. This epithelium rests on a layer of fibrous tissue, in which are numerous mucous glands, the ducts of one or two of which can be seen to pass up to the central lumen. Lying outside this stratum are three fragments of cartilage united by fibrous tissue and forming a fibro-cartilaginous layer surrounding the central structures for about 2 to 3 in. of their circumference.

REMARKS.

In discussing the origin of this teratoma a correct determination of the region in which it has arisen is necessary. As regards its situation, the tumour may be looked at from two points of view: either as lying in the renal area of the retroperitoneal region, or, on the other hand, as being connected with the region of the body axis; and, considering the intimate relation of the tumour to the spinal column, there is little doubt that it is correctly regarded as having arisen in the region of the body axis. Composite tumours arising in this region may be divided into two classes: Tumours derived from rudiments of tissue proper to the region itself, such as the myotomes, or immediately adjacent tissues which may become involved in the tumour formation, such as skin; and, secondly, tumours originating from duplication of the embryo. At the anterior and posterior extremities of the body axis, in which situations ectoderm and endoderm come into close connexion in the course of embryonic development, derivatives of all three germinal layers may be present in a tumour, whether the tumour has arisen by duplication or not. But in other parts of the axis, where no such connexion between ectoderm and endoderm occurs, the presence of a derivative of endoderm in a tumour, as in the present case, would indicate that the tumour had arisen through duplication of the embryo.

Teratomata of the anterior and posterior extremities of the axis are well known. Similar tumours occupying the peritoneal cavity, or situated in the retroperitoneal tissue in the neighbourhood of the kidney, have been described in the *Transactions of the Pathological Society* and elsewhere. But we have not been able to find the record of a case in which such a retroperitoneal tumour was intimately attached to the spinal column and actually occupied the spinal canal.

The Result of Double Oöphorectomy upon the Growth of the Uterus in the Rabbit.

By S. G. SHATTOCK.

IN the male sex one of the results following double castration, when this is carried out upon the young animal, is a hypoplasia or arrested growth of the accessory generative apparatus. In the case of the sheep, the observations of Dr. C. G. Seligmann and myself bearing upon this subject have been already recorded,¹ and are illustrated by dissections placed in the Museum of the Royal College of Surgeons. These preparations are fully described in the College catalogue, and I may quote the descriptions as the readiest way of stating what the results in question are:—

"No. 25A.—The bladder, with the urethra as far as the penile portion, together with the vesiculæ seminales and terminal portions of the vasa deferentia, of a Herdwick sheep which was castrated as a lamb in June, 1902, and killed in July, 1903. The vesiculæ are quite diminutive, not more than 1·7 cm. in length, and 7 mm. where widest; the terminal parts of the vasa deferentia exhibit a corresponding failure of growth, being less than half the normal diameter. There is a marked failure, moreover, in the development of Cowper's glands, these bodies, with investing musculature, measuring only 7 mm. in diameter. The urethra, though of full length, is of lesser diameter than in the intact, fully-grown ram, from a diminution in the thickness of its muscular wall. The condition of the prostate gland, which lies completely hidden between the muscular wall of the urethra and the urethral mucosa, is not shown."

In contrast with this, the vesiculæ of a full-grown intact sheep of the same breed measure 3 cm. in length, and in breadth, where widest, 15 mm., whilst Cowper's glands have a diameter of 15 mm.

In the sheep, as in the pig, the prostate gland, as already stated, lies hidden from sight between the urethral mucosa and the thick sheath of surrounding muscle, and for this reason the effects produced upon its growth by double castration are not easily recognized. In the dog, however, where the gland has a disposition similar to that in man, the hypoplasia following double castration, when carried out in the young, has been strikingly shown by Mr. C. S. Wallace in the figures

¹ *Proc. Roy. Soc. Lond.*, 1904, lxxiii, p. 49, and *Trans. Path. Soc. Lond.*, 1905, lvi, p. 57.

illustrating his communication on the "Results of Castration and Vasectomy upon the Prostate Gland."¹ *Mutatis mutandis*, it might be expected that the removal of the corresponding organ in the female, the ovary, would similarly be followed by a general hypoplasia of the sexual apparatus.

In the present communication the result of double oöphorectomy upon the growth of the uterus is alone considered. In the rabbit nothing can be deduced in regard to the pelvis, since there is no appreciable difference in the size of the normal pelves of the two sexes. A paper dealing with the result of oöphorectomy upon the growth of the pelvis in the cow will be submitted to the Pathological Section of the Royal Society of Medicine by Dr. C. G. Seligmann and myself.

In the case of the human subject the developmental correlation of these structures, the ovary and uterus, is by no means generally held; and it was for that reason that the following observations were carried out upon the rabbit. The positive results furnished by such direct observation must, I submit, outweigh in value the data furnished by cases of malformation or disease in the human subject, where doubt and misinterpretation may arise as to the sequence of events; for the ovaries, one or both, in whole or in part, may have once functionated and prematurely degenerated, and in such a case, should the accessory organs and passages have been fully developed, they would remain so, or would atrophy at a slower rate, and so bring about a disparity which might erroneously be read as evidence that the development of the ovary played no part in determining the growth of the accessory organs—a statement which is, indeed, at times made on such indirect grounds.

What is known in the case of the male in connexion with bilateral cryptorchism is so pertinent to this subject that I may here introduce it, for it might equally well be held in argument, from some such examples, that the testicles were not essentially concerned in the production of the external or secondary sexual characters in the male. In this condition the individual exhibits, in nearly all cases, the external characters of a well-developed male, but it is very rarely that fertility is recorded. In three cases cited by Curling in his classical work² there is a history of paternity. (1) A man, aged 29, married at the age of 20, had two children by his first wife, and had been married two years to a second. The penis was well developed and he had all the signs of virility; his testicles had not descended, and there was no scrotum.

¹ *Trans. Path. Soc. Lond.*, 1905, lvi, p. 80.

² "Diseases of the Testicle," 4th ed., 1878, p. 470.

(2) A man whose testicles had not descended had married twice, and had children by each wife, the virile functions being perfect. (3) A healthy labourer, aged 32, with the testicles in the inguinal canals. He was operated upon for strangulation of an oblique inguinal hernia of the left side. The left testicle was exposed during the operation; it was smaller than usual. He had a masculine development, was married, and his wife had had two children. Debrou's case (cited by Curling) is, however, more to the point, since in this a microscopic examination of the testes was made.¹ The patient, a man aged 42, was of ordinary stature, and the male characters were well marked. The right testis was retained in the inguinal canal; the left lay in front of the external abdominal ring. Death occurred from strangulated hernia. An examination of the testes (by Gosselin and Godard independently) showed no spermatozoa. Although this man had a son aged 8, his own testicles, at the age of 42, had become functionless; nevertheless, his external masculine characters had persisted.

The vast majority of double cryptorchids, however, are sterile, without being necessarily impotent. This is abundantly proved by Curling's own cases, in which the microscopic examination of the emission showed the absence of spermatozoa. The subjects, notwithstanding, presented proper external masculine characters: (1) Aged 38; externally had all the attributes of the male sex. (2) Aged 21; wore a moustache, and had abundant hair on the pubes; married twelve months. (3) Aged 19; vigorous, manly appearance, abundance of dark hair on the pubes. (4) Aged 30; in this case the pubic hair was scanty.

In the face of this, and other evidence, Curling was inclined to question the source of paternity in the three cases first cited. Mr. G. Bellingham Smith,² in an article upon this subject, states that he found mitosis very marked in two cases of undescended testicle (unilateral), and observes that, with the evidence supplied by Monod and Arthaud³ (which he accepts against Griffiths), it is not justifiable to deny paternity in any case in which a man, married young, is the subject of dispute; that the mass of evidence, nevertheless, is against the fertility of such subjects; that degeneration is found so early, being marked at the age of 20, that the testicles of a cryptorchid have probably a very short

¹ *Gaz. hebdom. de méd. et de chir.*, Par., 1861, viii, p. 3.

² *Guy's Hosp. Reports*, Lond. (1896), 1898, liii, p. 215.

³ In this case an undescended testicle was removed in an operation for strangulated hernia, the patient being aged 20. The organ was normal in size and aspect, and a microscopic examination of it revealed the presence of spermatozoa. *Arch. gén. de méd.*, Par., 1887, clx, p. 641.

period of activity. The only other case in which spermatogenesis has been proved in a cryptorchid is that by Beigel.¹ In this the presence of spermatozoa was discovered in the semen of a man, aged 22, both of whose testicles were retained in the inguinal region; the left was smaller than the right, but by no means wasted.

The point of this digression in relation to the present subject is that, although the undescended testicle may functionate sufficiently to bring about the appearance of the proper external masculine characters, the organ prematurely degenerates, whilst the male characters established by its function persist, the relationship between the two phenomena becoming in this way obscured. Corresponding facts may be cited from the lower animals. In the horse, as is well known, if castration is carried out in the foal (at the end of the first year), the fibro-fatty "crest" of the stallion fails to develop, the neck of the gelding coming to resemble that of the mare. If the adult stallion is castrated, the highly arched crest persists. In the horse, bilateral cryptorchism is of well-known occurrence in veterinary practice. Such animals, Mr. H. A. Woodruff informs me, have all the external characters of the stallion. If one testis only is descended, the cryptorchism being unilateral, the animal is castrated, in the usual course, as a gelding. It may afterwards grow restive and beget; the undescended testis may then require removal. This is quite a common event. These facts prove that in the horse the undescended testicle is not necessarily functionless. Such animals, nevertheless, are bad stock-getters: the undescended organs may have become functionless, although the animal remains potent.

The following instances are cited by Curling (*loc. cit.*, 4th ed., p. 30) from the "Memoirs" by Goubaux and Follin: (1) A horse, 12 years of age, though presenting all the characters of an entire animal, bore the marks of castration on the right side, but on the left there was no trace of cicatrix and no scrotal sac or testicle; erections took place in the vicinity of mares; after covering one, the fluid emitted from the urethra was examined and found to be destitute of spermatozoa. (2) After the purchase of a horse, 6 years old, a question arose whether the animal could be used as a stallion since no testicles were to be found and there were no marks of castration; he was allowed to cover a mare, but the examination of the fluid emitted on three occasions, at intervals of several days, afforded no trace of spermatozoa. These cases show that, although the animals presented the proper external or secondary sexual

¹ *Virchow's Archiv f. path. Anat.*, Berl., 1867, xxxviii, p. 144; cited by Bellingham Smith, *loc. cit.*

characters, the undescended testis was functionless at the time the mares were covered. Yet this fact cannot be taken as showing that the proper external male characters which were present had developed independently of the function of the testicles. Godart¹ also relates that a cryptorchid dog covered a bitch on heat four different times in March, 1856; the fluid ejaculated on each occasion was found destitute of spermatozoa. In February, 1857, this dog again covered a bitch; the sperm emitted contained no spermatozoa.

These observations correspond, then, with what is known of bilateral cryptorchism in the human subject; for in the great majority of cases this condition, as is universally allowed, is accompanied with sterility, though not with impotence, an examination of the emission showing an absence of spermatozoa. Such subjects may, nevertheless, as before stated, present all the external characters of maleness, and the penis may be normally developed. In the cases of sterility cited by Curling, in this connexion, it is essential to notice that the microscopic examination of the emission was not made at the initial stage of puberty, and that the absence of spermatozoa *at that date* cannot be substantiated. The results of castration in early life in preventing the appearance of the external, secondary male characters do not allow us to believe that these characters, when they appear, do so in conjunction with a functionless condition of the testicles. It still remains a matter for investigation whether the production of the external characters takes place independently of the spermatogenic function. That it results from the elaboration and circulation of an internal secretion may be accepted, but in my judgment it is probable that the internal secretion is not elaborated in the complete absence of spermatogenesis, the first of the two phenomena hanging in some way on the second.

There has recently come under my observation a case of sexual precocity in a cockerel which at first seems at variance with the accepted doctrine that the development of the external or secondary sexual characters is dependent upon the function of the testicle, but which, on analysis, is not so. Last September, Dr. H. C. Jonas sent to the College a chick, with the following history: The bird before it was two months old began to develop an enormous comb and wattles; when it was just over two months, the comb was about six times the size of that of a bird of the same breed and of the same age, and was of a vivid red colour. He crowed before he was three months old, and was in the habit of dropping his wing and running round the pullets in the

¹ "Études sur la Monorchidie et la Cryptorchidie chez l'Homme," Par., 1857.

manner of a sexually mature cock. The bird was sent up alive, and presented the ludicrous appearance of a small chick, without spurs or sickle feathers in the tail, but with the bright-red voluminous comb of a fully-grown cock. It was killed with chloroform on September 15, 1909. A dissection of the trunk showed that the testicles were altogether abnormal in size for the age. In the long axis they measured 2 cm., and in the transverse 1 cm. The histological examination of the right gland showed it to be completely matured, all the tubuli being of full size and spermatogenic to the extreme degree. The long, closely-looped vasa deferentia stood strongly out and were developed proportionally to the testicles. There was no enlargement of the adrenals. This may be particularly noticed, since in the human subject not a few examples of precocious puberty, in both sexes, have been found associated with adrenal overgrowth. The association, as brought out by the series of cases collected by Dr. Bulloch and Dr. Sequeira,¹ is so striking that Dr. C. G. Seligmann and I were induced to put it to an experimental test. The results, which were negative, are recorded in the *Proceedings of the Royal Society*.² The manner of the experiment was far from ideal, and to that degree it was inconclusive; for in the first place it was necessary to select an animal of which the male presented distinctive secondary characters, and as amongst mammals such are confined to the larger forms, we had to take a bird; and we chose the wild duck. But in selecting a bird another difficulty was forced upon us. Any influence which the adrenal may exercise upon sexual development is presumably due to the internal secretion of the cortex. And in the bird the cortical and medullary tissue of the adrenal are so intermingled as to render the isolation of the cortex for the preparation of an extract impossible. We were thus driven to use a mammalian adrenal, and chose that of the sheep, of which it was easy to obtain a supply. The regularly-repeated subcutaneous injection of a freshly-made salt-solution extract of the adrenal cortex into young wild ducks failed to accelerate the assumption of the male plumage, as compared with control birds. The injections invariably produced a local (aseptic) necrotic knot in the subcutaneous fat, but no result of any general kind was detectable.

The examination of the testicles of a control cockerel hatched from the same batch of eggs, and killed on September 21, 1909, a week later than the other, showed that in the longer vertical direction

¹ *Trans. Path. Soc. Lond.*, 1905, lvi, p. 189.

² S. G. Shattock and C. G. Seligmann, *Proc. Roy. Soc. Lond.*, 1908, Ser. B., lxxx, p. 473.

they measured no more than 6 mm. and not more than 4 mm. in the transverse. The spurs and sickle feathers of the tail were undeveloped, as was also the comb. In the case of the precocious cockerel, although the testicles are fully matured and functioning, the comb is the only element of the secondary or external characters that has developed; neither the spurs nor the sickle feathers have as yet appeared.

The real test as to which secondary characters are to be accounted as sexual and which not, is furnished by the results that follow castration or oöphorectomy in the young.

It is certainly the common belief that the spurs are organs indicative of sexual, as distinguished from the general, maturity of the male bird, and the same of the sickle feathers of the tail. Yet that this is not so is shown by the fact that these particular external characters appear after the castration of the young bird. The capon is furnished both with spurs and sickle feathers; what it does not grow is the comb. The precocious cockerel, therefore, offers no new problem in this regard. The comb is voluminous and masculine; the spurs and sickle feathers, which are marks not of sexual but of general maturity, have not yet appeared: the bird is precocious in its sexual development only.

The Results of Double Oöphorectomy upon the Growth of the Uterus in the Rabbit.

The animals used for the following observations were quite young, and the operation was carefully carried out under an anæsthetic, the abdomen being opened in the mid-line, and the parts fully exposed by turning aside the intestines, without being themselves injured or forcibly raised and displaced; the ovary was seized with a fine pair of forceps and cut away. No ligatures were used for any purpose; there was no hæmorrhage. One animal was operated upon on October 12, 1908; a second on November 5, 1908. At the dates of operation they were of the same size; nothing untoward followed. On April 10, 1909, one of the two died. As the animal was not fully grown, the final effect of the oöphorectomy upon the growth of the uterus is not in this case determinable.

The other animal was killed on October 11, 1909, a year after the operation, and the pelvic viscera have been permanently preserved in the Museum of the Royal College of Surgeons. It weighed before being killed 3 kilos 280 grm., and had remained throughout the time in excellent condition. On opening the abdomen the unnatural tenuity of

FIGURA 1.

Cuniculi adulti (pondere 3 kilos 280 grm.) monstrantur uteri, dexter et sinister, oyariis dum juvenescebat excisis.

Uteri qui in summâ vaginâ transverse disponuntur insigniter tenuantur.

A—Uterus sinister.

B—Fallopîi tuba.

C—Tubae terminatio cicatrizata, extremitate fimbriatâ cum ovario amputatâ.

Magnitudinis naturalis.

The uteri with the highest part of the vagina of a fully-grown rabbit from which the ovaries had been removed when the animal was quite young. The uteri, which are disposed at right angles to the long axis of the vagina, are remarkably slender and undergrown. The uterus, A, passes near the spot marked B into a still finer and more tortuous Fallopian tube, which terminates in a rounded cicatrized stump, C, the fimbriated end having been removed with the ovary.

The animal weighed 3 kilos 280 grm. Natural size.

FIGURA 2.

Cuniculi adulti integri (pondere 3 kilos 200 grm.) monstratur uterus sinister, ut cum illo cuniculi præcedentis comparetur. Partum est animal mense Augusto, 1909; interfectum est mense Novembre, 1909.

A—Uterus sinister.

B—Fallopîi tuba.

C—Extremitas tubae fimbriata.

D—Ovarium.

Magnitudinis naturalis.

The left uterus of a fully-grown rabbit, weighing slightly less than the preceding—viz., 3 kilos 200 grm., showing the markedly greater size of the organ. The animal had been pregnant, for the first time, in August, 1909, and was killed November 8, 1909.

A—Left uterus.

B—Fallopian tube.

C—Fimbriated extremity of the tube.

D—Ovary.

Natural size.

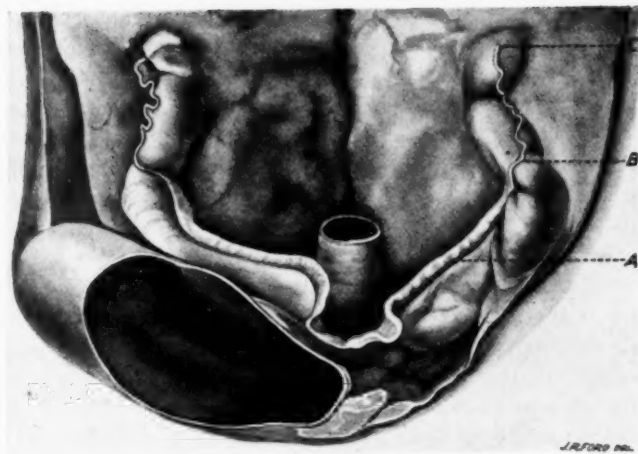


FIG. 1.

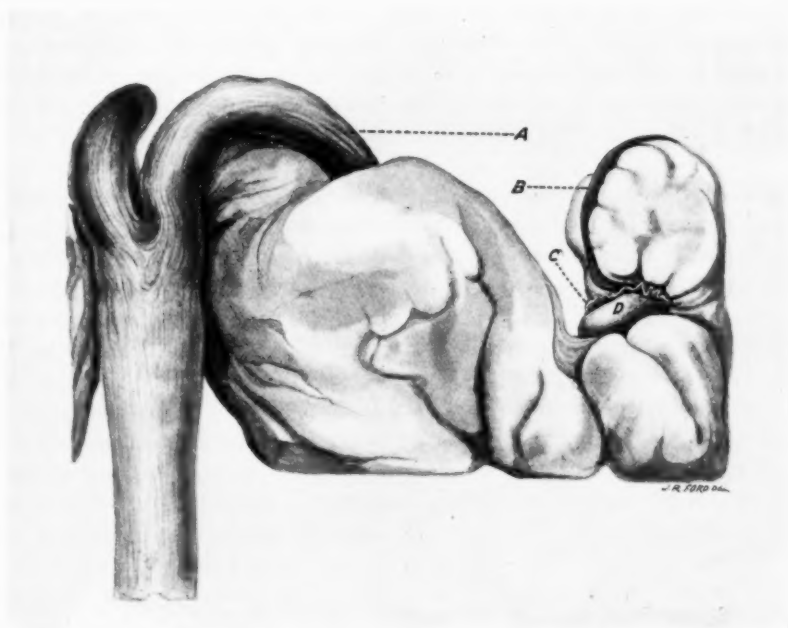


FIG. 2.

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the uteri (which are disposed horizontally at the summit of the vagina) at once arrested attention, the hypoplasia of the organs being obvious at the first glance. Each uterus, which is so thinly walled and flaccid as to assume the form of a narrow flattened band instead of being, as it normally is, stiff and cylindrical, measures in breadth across the flatter side 2 mm. The length of each, as taken with a fine thread laid along its curves, is 4.5 cm. At its termination it merges into a still finer and more tortuous Fallopian tube; this, as measured along its coils, is 2 cm. Each tube terminates in a rounded stump, the fimbriated end having been removed with the ovary.

For comparison I may give the measurements of the corresponding parts in a fully-grown rabbit, of less weight than that from which the ovaries had been removed, and which had been once pregnant. The rabbit in question weighed shortly before it was killed 3 kilos 200 grm.—i.e., 80 grm. less than that spayed. The animal had young for the first time in August, 1909, and was killed on November 8, 1909. The diameter of each uterus is 9 mm. The length of each, as taken along its curves, is 9 cm. The length of the Fallopian tube along its curves is 14 cm.; the diameter of the tube is 2 mm.

A comparison of the uteri from the oöphorectomized rabbit with those of the intact will show the marked undergrowth of the organs resulting from the removal of the ovaries, and prove that the sexual gland, as in the male, dominates the growth of the rest of the generative apparatus.

There is a traditional statement of a converse kind current amongst poultry-breeders, to the effect that division or partial removal of the oviduct in young hens is followed by want of development of the ovary. The object sought by the proceeding is, as in castration of the cockerels, to render the birds sexless and bring about their fattening. Although, from a scientific standpoint, such a result would be so unlikely, it is widely believed to follow, as may be seen by a reference to Mr. W. B. Tegetmeier's book on "Poultry" (3rd ed., 1898, p. 131). The fallacy of the belief Dr. C. G. Seligmann and I have proved by direct observation. The removal of the duct was carried out upon young birds which had not arrived at sexual maturity. As they grew up nothing was discoverable in their external characters by which they could be differentiated from intact control hens of the same breed. A dissection after they had reached maturity showed that the ovary was fully developed, with large protuberant yolks.

Pathological Section.

November 16, 1909.

Systematic Histological Examination of the Central Nervous System of a Case of Transverse Lesion of the Spinal Cord in the Lower Cervical Region.

By SIDNEY V. SEWELL and H. HUME TURNBULL.¹

T. T., AGED 14, suffered from a fracture-dislocation of the spinal column in the mid-cervical region, as the result of a motor-car accident. The patient was admitted to Charing Cross Hospital under the care of Mr. Stanley Boyd, and was seen by Dr. F. W. Mott, to whom we are indebted for the opportunity of examining the central nervous system, and for the following brief notes of the case:—

Shortly after the accident there was found to be complete flaccid paralysis of the trunk and limbs, breathing being wholly diaphragmatic, and the only movements which remained were slight flexion and pronation of the forearms. All the deep reflexes were absent, and the plantar response was at first flexor, and later doubtful extensor in type. Later he recovered some movements in the right arm and slight voluntary flexion of the thighs, which was accompanied by spasticity of the lower limbs. There was at first marked comparative loss of all forms of sensation in the legs, with almost total loss in the trunk up to the level of the first rib, but ultimately sensation improved considerably, with the exception of the thermic sensibility, which remained very markedly impaired. For some days after the accident there was incontinence of urine and faeces, followed later by persistent retention, and priapism

¹ From the Pathological Laboratory of the London County Asylum, Claybury, Essex.

was a marked feature throughout. The patient died eight weeks after the accident.

The brain-stem and spinal cord were hardened in Müller's fluid for a period of five months, while the cerebellum, mid-brain, and cerebral hemispheres were in 5-per-cent. formalin solution for the same period. Sections from the brain-stem and spinal cord were stained by (1) Marchi's method; (2) Weigert's and Weigert-Pal's method; (3) Hæmatoxylin and Van Gieson's stain. The sections stained by Weigert's method and Pal's modifications showed practically no changes, and those described were seen in the sections stained by the Marchi method. Sections from the mid-brain, cerebellum, and cerebral cortex were stained by (1) Marchi's method; (2) Nissl's method.

It is proposed to discuss, firstly, tract degenerations ascending and descending from the lesion, which was found to be situated at the level of the fifth cervical segment; and, secondly, the changes resulting therefrom in the cells of origin of the injured fibres.

DESCENDING DEGENERATIONS.

In the anterior columns: A well-defined tract on either side of the anterior median fissure was found to be degenerated as low down as the third sacral segment. The degeneration in this situation was almost as well marked in the region of the fourth lumbar segment as in the dorsal region, but below the lumbar region the number of degenerated fibres rapidly diminished, and none could be observed below the third sacral segment. Well-marked degenerations of a somewhat scattered nature were also present in the region of the rubrospinal and Deiters' descending tracts, a few degenerated fibres being still present at the level of the third sacral segment (*vide* figs. 1—6). Very marked degeneration was present in the crossed pyramidal tracts throughout (*vide* figs. 1—6).

Posterior columns: Well-marked degenerations of the endogenous descending tracts of the posterior columns were present. The comma tract was degenerated as low as the ninth dorsal segment, while below this region the septo-marginal tract was degenerated as far as the third sacral segment, where only a few degenerated fibres, situated quite superficially on either side of the posterior fissure, were to be observed. Flechsig's oval area was very definitely degenerated below the ninth dorsal segment, and could be seen as far down as the last lumbar, but not in the sacral segments. In the dorsal region many degenerated



FIG. 1.
Third sacral segment.



FIG. 4.
Seventh dorsal segment.



FIG. 7.
Sixth cervical segment,
level of lesion.



FIG. 2.
Fourth lumbar segment.



FIG. 5.
Third dorsal segment.



FIG. 8.
Fourth cervical segment.



FIG. 3.
Ninth dorsal segment.

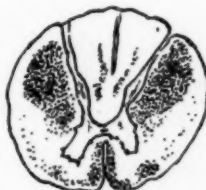


FIG. 6.
Second dorsal segment.

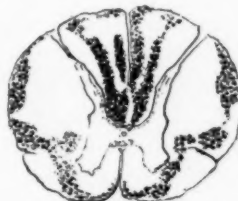


FIG. 9.
Third cervical segment.

Sections of the spinal cord at various levels to show the degenerated fibres stained by Marchi's method. (Drawn with Edinger's projection apparatus.)

fibres were present close to the grey matter in the deeper portion of the columns. Degenerated fibres were noticed within the grey matter, especially in the region of the anterior horns, while the posterior portions of the grey matter were comparatively free (*vide* figs. 1—6).

Level of the lesion, fifth and sixth cervical segments: The degeneration was extremely diffuse throughout all regions of the cord, though less marked in the deeper portions of the columns of Goll than elsewhere. On the whole, however, the regions of the efferent tracts showed a more intense degeneration than those of the afferent tracts. The grey matter also showed considerable degeneration. Many degenerated fibres were present in the posterior roots, and could be seen proceeding thence in the direction of the anterior horns. The grey commissures showed degeneration, which was especially well marked in the anterior commissure. Great numbers of degenerated fibres could be seen sweeping out from the region of the anterior horn cells. Sections stained with hæmatoxylin and Van Gieson's stain showed an intense inflammatory reaction, slighter in the white matter, but very intense in the grey matter, where the vessels were dilated and surrounded with large collections of round cells, and in many places tiny capillary hæmorrhages could be seen (*vide* fig. 7).

ASCENDING DEGENERATIONS.

In the posterior columns a very intense degeneration of the fibres of the columns of Goll and Burdach was present, more especially of those of the deeper portions, while the root zones and the region of the comma tract were comparatively free. Above the sensory decussation the mesial fillet still showed a considerable number of scattered degenerated fibres (*vide* fig. 12). The combined fillet in its passage through the mid-brain showed a much greater degeneration owing to the large number of degenerated fibres which had entered it from Gowers' tract. The direct cerebellar tracts appeared entirely degenerated, and could be traced through the restiform bodies, of which they formed the superficial dorsal portion, into the cerebellum, the degenerated fibres going partly into the middle lobe, and partly into the lateral lobes. The greater number of these degenerated fibres could be traced up to the granular layer of the cortex, but none were seen among the cells themselves, while others, much fewer in number, appeared to pass directly to the neighbourhood of the cells of the dentate and roof nuclei.

Such a distribution of the fibres entering the cerebellum from the cord agrees with the results obtained in animals by Horsley and Clarke [3]. By this arrangement the effector cells of the cerebellum—i.e., those of the dentate and roof nuclei—are placed in a similar position to those of the cerebrum—i.e., the Betz cells of the ascending frontal convolutions—both receiving impulses from the afferent system as well as from the association areas of their respective cortices.

The antero-lateral ground bundles showed well-marked degeneration of Gowers' tracts, which were traced upwards through the brain-stem

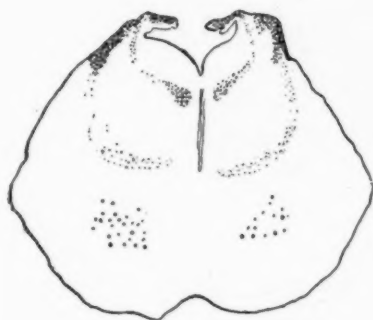


FIG. 10.
Section through upper part
of pons.



FIG. 12.
Section through medulla,
mid-olivary region.

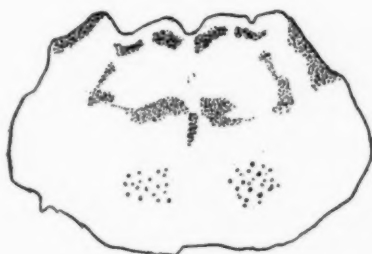


FIG. 11.
Section through mid-pons.



FIG. 13.
Section through medulla at level
of pyramidal decussation.

Sections through the brain-stem, showing degenerated fibres stained by Marchi's method.

to the upper portion of the pons, giving off as they ascended a large supply of fibres to Deiters' nucleus. In the upper pontine region the tracts subdivided, one portion passing by means of the superior cerebellar peduncle into the cerebellum, while the remainder joined the fillet. A considerable amount of degeneration was also observed in the anterior ground bundles, and this could be traced through the brain-stem in the continuation of these bundles in the posterior longitudinal bundles. Of these degenerated fibres in the posterior longitudinal bundles many passed to the region of Deiters' nuclei, others into the valve of Vieussens, and the remainder largely into the corpora quadrigemina. Longitudinal sections showed large numbers of degenerated fibres sweeping from the mesencephalon to the lateral nucleus of the thalamus.

Well-marked degeneration in the grey matter of the cord could be observed for a distance of three segments above the lesion, the degenerated fibres sweeping across the anterior commissure. A slight amount of retrograde degeneration was seen in the pyramidal tracts above the lesion, but only a few fibres were affected.

CELL CHANGES.

The cells of the grey matter of the spinal cord appeared quite normal, with the exception of those of Clarke's columns. Here there were very marked changes, most of the cells being globular with absent or excentric nuclei, and marked diminution of the tigroid substance. None of the cells of these columns appeared to be normal, but the degree of degeneration showed considerable variability (figs. 14—15).

In Deiters' nuclei many of the large cells showed considerable change, being swollen and irregular in shape. Their nuclei were excentric, and more or less marked chromatolysis was present (fig. 14, 3). On the other hand, some few of the large cells and all the small cells were quite normal in appearance.

The cells of the red nuclei seemed to be distinctly diminished in number, and for the most part the large cells present were very irregular in shape and the nuclei were excentric and pale, the cell-body staining poorly by Nissl's method and exhibiting marked chromatolysis. Satellite cells could be seen in many cases in juxtaposition to the irregular cell-walls, and the position of these cells distinctly suggested that they were acting as phagocytes. The appearance presented by these cells of the red nucleus was in striking contrast to that of the normal cells of the third nerve nucleus in the same section (fig. 14, 5 and 6).

The cells of the locus niger were affected in varying degrees, many being perfectly normal while some showed definite chromatolysis and nuclear excentricity (fig. 14, 4).

Sections of the central convolutions taken from the motor area of the cerebrum, as shown in the figures, were stained by Nissl's method and by modifications of this method—e.g., polychrome blue.

Section I, from the mesial surface of the leg area (fig. 15), showed in all nineteen Betz cells, only three of which can be described as quite normal in appearance, the remainder presenting every degree of chromatolysis, from slight perinuclear involvement to total disappearance of the Nissl bodies throughout the entire cell.

Some few of the cells persisted as mere irregular ghosts, taking practically no stain and presenting no nucleus, while the majority showed considerable excentricity of the nucleus, with appearance of vacuolation and a moderate degree of chromatolysis. The cells presenting marked changes had in many cases "satellite cells" lying in irregularities of their outline. The large pyramidal cells appeared quite normal, and no changes could be observed in the post-Rolandic region.

Section II (*vide* fig. 16): Twenty-four Betz cells were counted, nine of which were practically normal in appearance. One definite ghost cell was seen, and the remaining fourteen cells showed varying degrees of chromatolysis and nuclear excentricity. Again the large pyramidal cells and the cells of the ascending parietal convolution appeared quite normal, and this was the case in all the sections examined.

Section III, from the upper arm area (*vide* fig. 16), shows eleven Betz cells, eight of which were normal, the remaining three showing definite chromatolysis and nuclear excentricity.

Section IV, from the lowest part of the arm area (*vide* fig. 16): Eight cells were counted, three of which were fairly normal, while the remaining five showed excentricity of the nucleus and a considerable amount of chromatolysis.

Section V (*vide* fig. 16): Here the Betz cells are considerably smaller than in the other sections, and of six cells counted, five were quite normal, while one showed slight perinuclear chromatolysis.

Section VI (*vide* fig. 16): The Betz cells, of which six were seen, were still smaller in size, and all appeared normal.

Section VII, face area: Four cells were seen, all of which were normal.

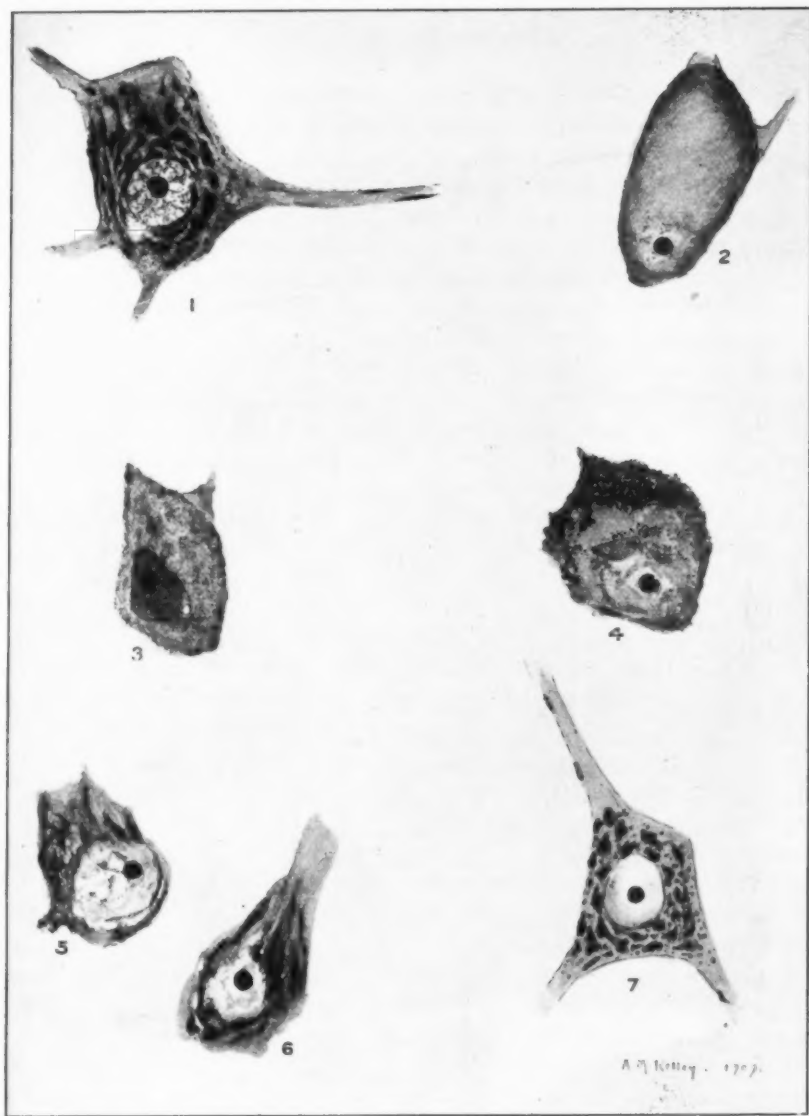


FIG. 14.

Cells from spinal cord and brain-stem. (1) Anterior horn cell, normal, and (2) cell of Clarke's column, showing marked chromatolysis and excentricity of nucleus. These cells are from the same section of the fifth dorsal segment, and show the contrast between the degenerated cells of Clarke's column below the lesion and the normal cells of the anterior horn cell similarly stained. (3) Cell of Deiters' nucleus, showing chromatolysis and nuclear excentricity. (4) Cell of locus niger exhibiting similar changes. (5 and 6) Cells of red nucleus, showing varying degrees of chromatolysis and excentricity of nucleus. (7) Normal cell of third nerve nucleus from same section as (5 and 6), to show contrast between the cells of this nucleus and those of the red nucleus.

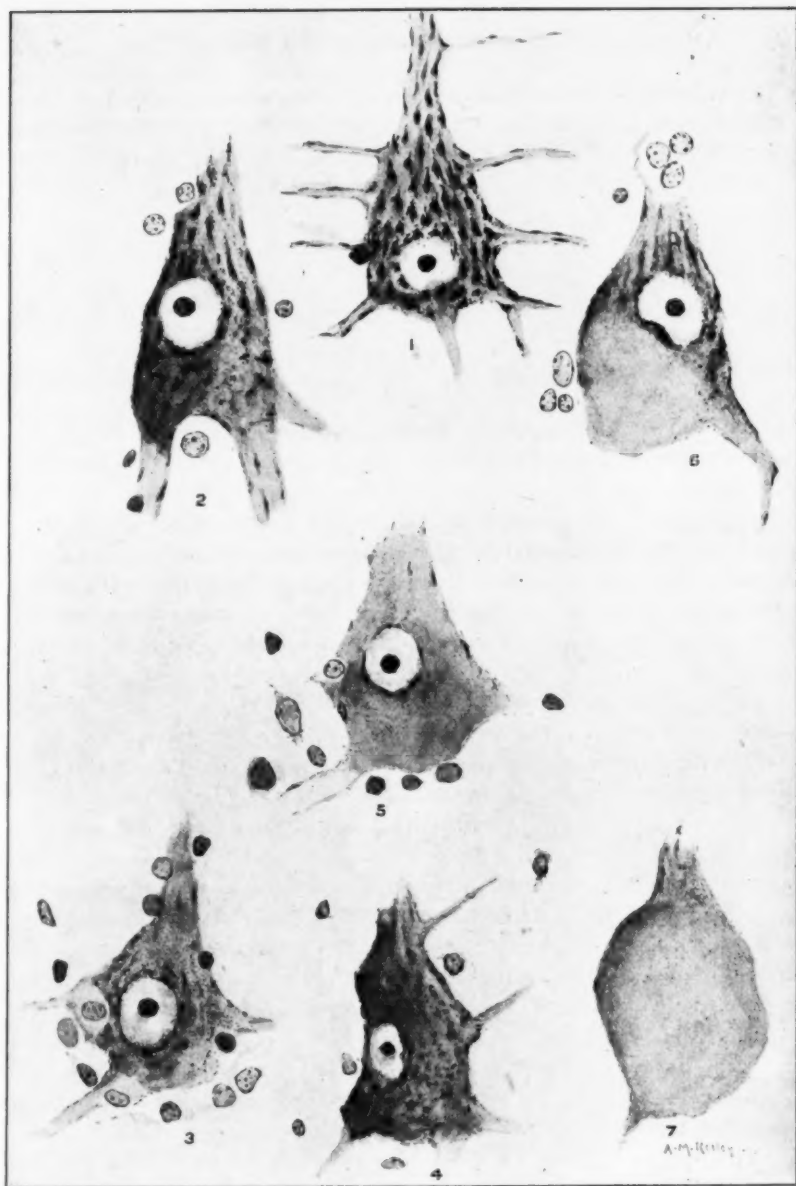


FIG. 15.

Betz cells from motor area of cortex, showing all stages of chromatolysis and nuclear excentricity from (1) the normal cell to (7) a cell with completely extruded nucleus, the cell being swollen and showing no trace of tigroid substance. Satellite cells are seen around many of the Betz cells.

The above description gives an account of an average section of each of these areas, and summarizes the results obtained from the examination of a large number of sections in each case. The changes in the cortical cells of the two hemispheres were similar.

COMMENTS.

Two points seem worthy of special mention in connexion with the degenerations described :—

(1) The definite tract degeneration which was present on either side of the anterior median fissure as far down as the third sacral segment. This tract was well marked down to the level of the fourth lumbar segment, but the degenerated fibres rapidly lessened in numbers in the sacral cord, had almost entirely disappeared by the time the third sacral segment was reached, and could not be recognized below this. The direct pyramidal tract is usually described as ending at the lower dorsal or first lumbar segment, and we regard the degenerated fibres seen in our case as representing the tract originally described by Mott [4] as descending the cord from Deiters' nuclei or cranial motor nuclei.

(2) It is of very great interest to notice the large direct afferent supply from the limbs received by Deiters' nucleus by means of its connexions with Gowers' tract and the fibres ascending from the cord in the posterior longitudinal bundles (*vide* fig. 17).

In this respect our results differ considerably from those of Fraser [1], who, in an experimental inquiry into the results of lesions in the posterior longitudinal bundle of animals, was able to obtain only slight ascending degeneration from the cord to Deiters' nucleus, and concluded that the supply of afferent fibres from the cord to Deiters' nucleus in these bundles was insignificant. Such large afferent connexions as we found, when considered together with the fibres coming to Deiters' nucleus from the vestibular nerves and from other important cranial nerves by means of the posterior longitudinal bundle, place that nucleus in an excellent position to act as a great co-ordinating and equilibrating centre, as has been so often suggested.

The reasons why the cells of the central cortex show such varying amounts of change are rather difficult of explanation. It has been suggested by Dr. Mott that the real reason of this variability lies in the fact that the changes present in a cell after such a lesion are proportional to the amount of injury done to the total neurone. If a

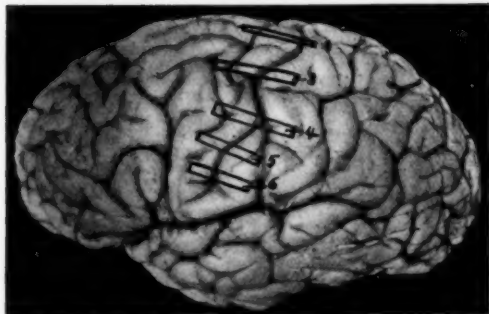


FIG. 16.

Showing the parts of the cortex from which the sections were made. Section 1, on the mesial surface of the leg area, is not shown, nor is section 7 from the face area. Similar sections were taken from the opposite hemisphere.

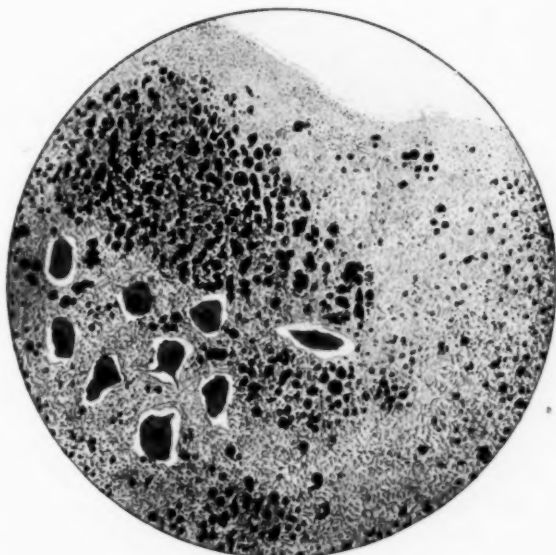


FIG. 17.

Showing degenerated fibres round cells of Deiters' nucleus (semi-diagrammatic).

neuraxon has already given off a large number of collaterals above the place of injury, then the changes in the cell will be only transitory and the cell will in all probability completely recover. This would explain the fact that the cells of Clarke's column presented fairly uniform changes, while the cells of the cortex and of Deiters' nuclei presented a varying amount of change, as the degeneration would depend inversely on the number of collaterals given off from the axons of the cells above the seat of injury. On the other hand, there was a very slight amount of movement in the thigh and some movement in the arms after the accident, so that it is probable that at least some of the fibres of the pyramidal tracts escaped injury.

Our observations are in entire agreement with the results obtained by Gordon Holmes and Page May [2], in that the only cells of the cortex we found affected were the large Betz cells of the ascending frontal convolutions, and the large pyramidal cells were everywhere perfectly normal.

In conclusion, we wish to thank Dr. Mott for his kindness in giving us the material and for his help in the work.

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A Method for Studying the Action of Blood-fluids and other Substances on the Leucocytes.

(Preliminary Contribution.)

By HELEN CHAMBERS.¹

THE method described in this paper originated in an endeavour to find some means of testing whether the blood-fluids of cases with marked leucocytosis contain substances exerting an attractive influence on the leucocytes.

In November, 1908, Dr. Constant Ponder² described a method for the examination of living leucocytes *in vitro*. He takes a small roll

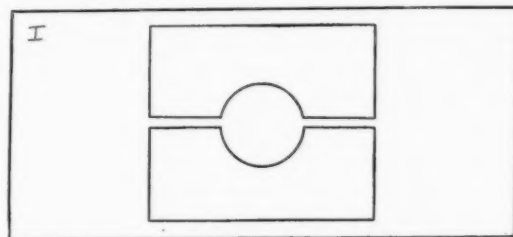


FIG. 1.

of plasticine and places it on a slide in such a shape as to form a cell with one aperture. The cell is next filled with fresh blood, a coverslip is placed over it and is pressed down by means of a glass slide until the coverslip comes in contact with the blood. The whole apparatus is then incubated at 37° C. in normal saline solution, and after the blood has clotted the leucocytes are found in large numbers collected on the slide and on the under surface of the coverslip.

The above method has been modified as follows: Instead of using a plasticine cell a glass slide has been made for me by Mr. C. Baker, of Holborn, similar to that shown in fig. 1. It contains a central cell 12 mm. in diameter and 1 mm. in depth, with an exit and entrance aperture on either side 1 mm. wide. The cell is surrounded by a raised

¹ From the Cancer Research Laboratories of the Middlesex Hospital.

² *Proceedings of the Cambridge Philosophical Society*, 1909, xv, pt. 1, p. 30.

platform, on which a coverslip can rest. To use the slide, a coverslip is placed on it in such a way that it forms a roof for the cell and has one edge projecting slightly from one side of the platform, the latter arrangement being in order to facilitate subsequent removal of the coverslip.

A drop of melted paraffin wax (melting point 40° C.) is placed in the centre of one edge of the coverslip, and a second drop in a corresponding position opposite to this; the paraffin being placed over the centre of the sides of the cell which are complete, and not over either aperture. The slide is next warmed, and as the paraffin melts it runs in between the coverslip and slide, and if a small quantity only has been used, it is limited by the edge of the cell and the margins of the openings on either side. The slide is allowed to cool until the paraffin solidifies. Two small bars of paraffin are next placed on either side of one aperture so as to serve as a guide to fluid entering the cell and prevent it spreading laterally. A drop of fresh blood is placed at the outer end of this opening, and the slide is raised slightly at this end. The fluid runs in by capillary attraction, more drops of blood are added, and with slight manipulation the whole cell is easily filled without air bubbles. It is next incubated at 37° C. in a covered flat dish containing a few drops of water, but not sufficient to reach the upper surface of the slide. The cell is hence surrounded by an atmosphere saturated with water vapour.

If it be desired to test the effect of any substance on the leucocytes, a film of the material to be tested is made on half the coverslip by means of the edge of a glass slide passed from the centre of the coverslip to one end in the same way that blood-films are made. The film is allowed to dry in air, and in the case of micro-organisms is fixed by heat. The coverslip is next turned over on to the cell, so that half the roof is covered with the material to be tested and the other half is clean. Each slide is numbered at one end, and I make it a matter of routine to turn the "film" portion of the coverslip towards this end of the slide. For further security that portion of the coverslip on which the film has been spread is marked with two drops of sealing-wax.

The time of incubation in most cases has been three-quarters of an hour. After removing the slide from the incubator the coverslip is raised, is washed in warm saline solution, and the leucocytes are then fixed and stained by any of the ordinary methods for staining blood-films, and a permanent preparation is thus obtained.

The results vary according to the condition of the coverslip, and are as follows:—

(1) *The Coverslip is Clean.*—The distribution of the leucocytes is comparatively uniform and the coverslip has the appearance shown diagrammatically in fig. 2.

(2) *A Film is made on Half the Coverslip with an Emulsion of Micro-organisms in Distilled Water.*—The leucocytes collect in large numbers on the film half, and are relatively scanty on the opposite half. Fig. 5 shows a microphotograph after using an emulsion of typhoid bacilli. It will be seen that the leucocytes tend to collect in largest numbers along the central line, probably because the organisms are more numerous here than elsewhere. The organisms which have been tested are: *Staphylococcus aureus* and *albus*, *Pneumococcus*, *Micrococcus tetragenus*, *Bacillus tuberculosis*, *Bacillus prodigiosus*, *Bacillus typhosus*, *Bacillus coli* and *Bacillus anthracis*. The results in all

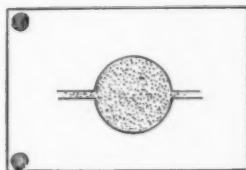


FIG. 2.

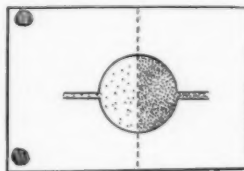


FIG. 3.

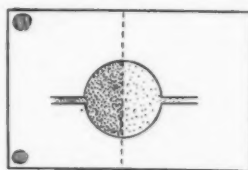


FIG. 4.

cases have been similar. In the case of *Bacillus anthracis* virulent and non-virulent cultures have been used. A thick emulsion of virulent anthrax bacilli was made from a sub-culture taken directly from the spleen of a mouse which died in eleven hours from septicæmia. The leucocytes were markedly attracted.

(3) *A Film is made on Half the Coverslip with Blood Serum.*—The blood is collected in a Wright's tube, is allowed to clot, is centrifugalized, and the clear serum pipetted off. In a series of thirty experiments the results in all cases are the same—viz., the leucocytes collect on the clean side of the coverslip and are extremely scanty on the film side, shown diagrammatically in fig 3. The blood serum of five normal persons has been tested, as well as two sera from cases of typhoid fever and pneumonia respectively. Rabbit's serum has a similar action on human white corpuscles.

(4) *A Film is made on Half the Coverslip with Blood Plasma.*—The results are not so uniform as when using serum. In a series of forty experiments, in twenty-two the leucocytes were attracted to the film half, and in eighteen the distribution was uniform (fig. 2 and fig. 4). The plasma was obtained by mixing 4 volumes of blood with 1 volume of varying strengths of sodium-citrate or potassium-oxalate solution. The mixture was centrifugalized and the fluid pipetted off. The results indicate that if the plasma be obtained rapidly, and with as little destruction of the blood-corpuscles as possible, the leucocytes in the cell (which are themselves bathed with serum) are attracted to the plasma. If, on the other hand, the plasma be left in contact with the corpuscles, and especially if there be also present a salt like potassium oxalate, which assists in the destruction of the cells, the plasma acquires properties like those of serum and repels the leucocytes. The eighteen cases in which the distribution of the leucocytes was uniform seem to be explained by supposing the plasma in an intermediate condition—i.e., as containing some products of breaking-down cells, but not sufficient to cause repulsion.

In connexion with the possibility that it is the products of broken-down blood-corpuscles (whether the red or the white cells the experiments do not show) which act as the inhibitory factor to the leucocytes, the results of testing the serum from the three following cases are of interest: (1) A pleural effusion of tuberculous origin; (2) an ascitic fluid from a case of chronic peritonitis; (3) the exudation from an oedematous ovarian fibroid. All the fluids were clear—contained very few cells and clotted well spontaneously. In the case of the pleural effusion the distribution of the leucocytes was uniform; in the other two cases the leucocytes were attracted. Thus the "serum" in these exudations acted like blood plasma. It is possible that this result is explained by the fact that the fluids contained comparatively few cells, and hence that very little destruction of cells could occur in the process of clotting.

The explanation of these results is difficult. In order to eliminate the action of the salt in the cases where plasma was used, two experiments were made as follows:—

Experiment I.—A film was made on half the coverslip with a mixture consisting of 4 volumes of serum and 1 volume of a 20-per-cent. solution of potassium oxalate in distilled water. The leucocytes acted as if serum alone had been used, and were collected in large numbers on the clean half of the coverslip and were scanty on the film half.

Experiment II.—A film was made on half the coverslip with the clear fluid obtained after centrifugalizing a mixture of 4 volumes of fresh blood and 1 volume of a 20-per-cent. solution of potassium oxalate in distilled water. The leucocytes were markedly attracted to the film side. From this it follows that the result is not determined by the potassium oxalate.

In order to determine whether surface tension has any marked influence, a film consisting of a mixture of ground glass and blood serum was spread on half the coverslip. The leucocytes in this case acted

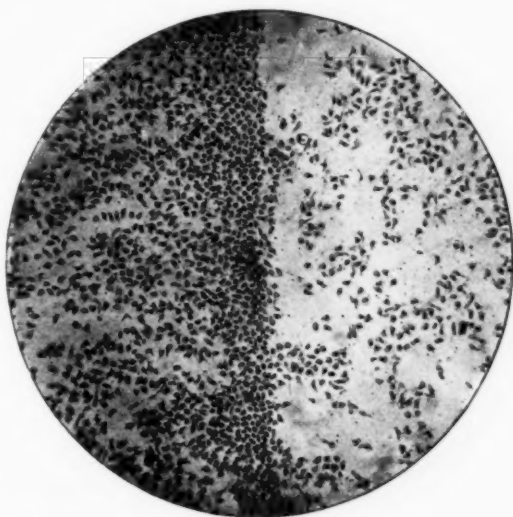


FIG. 5.

as if serum alone had been used and adhered in large numbers to the clean half of the coverslip, being very scanty on the film half; hence the roughening of the surface caused by the ground glass appears to have no effect.

Microscopic sections of the clot show that it contains very few leucocytes. The clot seems to act mechanically as a platform keeping the leucocytes in close contact with the coverslip. This is indicated by the fact that if citrated blood be used to fill the cell, the leucocytes sink rapidly owing to the action of gravity, and very few are found to adhere to the roof.

78 Chambers: *Action of Blood-fluids, &c., on the Leucocytes*

An emulsion of washed leucocytes, obtained by mixing pus with saline solution, centrifugalizing, and washing repeatedly, has been used to fill the cell. The slide in this case is reversed, incubated, and later the coverslip is removed and washed in saline solution. If the leucocytes are dead, none adhere to the coverslip. If alive, a distribution of leucocytes similar to that obtained with blood is found to occur according as either micro-organism or serum is used for making the films.

In all these experiments the film of leucocytes is readily seen with the naked eye when unstained, and the sharp line of demarcation between the two halves in cases of marked attraction or repulsion can easily be made out. The leucocytes are very irregular in shape. All kinds are to be found—except basophile cells, which I have not yet seen. No substance used up to the present has shown any special attractive action for leucocytes of one variety as compared with the others. I have seen nothing to indicate that in those cases in which the leucocytes are absent from the film half they have previously adhered and have subsequently broken down. It seems rather that they remain in the underlying serum, where they can be found.

Pathological Section.

January 18, 1910.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

An Experimental Investigation into the Origin and Cause of the "Pancreatic" (Cambridge) Reaction in the Urine.

By P. J. CAMMIDGE and H. C. G. SEMON.

IN a paper read before the Royal Medical and Chirurgical Society on March 13, 1906, by one of us, a method of examining the urine in suspected cases of pancreatic disease was described, and the results of 100 examinations of specimens from 94 persons tabulated. These showed that, while the urines of normal individuals and those in which there was no reason to suspect disease of the pancreas gave no reaction, specimens from patients who, clinically and at operation, showed evidence of inflammatory affections of the gland gave a characteristic crystalline deposit. Further experience with over 1500 specimens, histological examination of the pancreas in 51 cases where the urine had been examined during life, and the published results of other observers have served to confirm these conclusions and to show, as E. H. Goodman¹ has said, that, "taken in connexion with the clinical history and an examination and careful study of the fæces, the Cambridge reaction is strongly suggestive of inflammation of the pancreas."

Chemical investigations of the products obtained in this urinary reaction have indicated that it is due to a sugar-complex, probably a nucleoglycoprotein, which on hydrolysis with hydrochloric acid gives rise to a substance having the reactions of a pentose,² and that the typical

¹ *Annals of Surg.*, Philad., 1909, xlix, p. 183.

² *Proc. Roy. Soc.*, 1909, B, lxxxi, p. 372.

crystals obtained in the reaction are pentosazone crystals. This fact lends support to the view that the reaction is dependent upon degenerative changes in the pancreas, for, according to Gründ,¹ the percentage of pentose in the dry weight of the pancreas is nearly five times as great as in any other organ of the body, and, according to Blumenthal,² it is more loosely combined and more readily set free than the corresponding sugar in other tissues.

We have recently performed a series of experiments upon dogs, with the object of discovering whether the reaction was in reality associated with diseases of the pancreas, and, further, if so, whether it was dependent upon degenerative changes in the tissues of the pancreas itself or was consequent upon metabolic disturbances arising from interference with the functions of the gland. The operative portions of the work were carried out by one of us (H. C. G. Semon) in the pathological department of the University of Freiburg, and the chemical investigations conducted by the other (P. J. Cammidge) in London. Five dogs were operated on in all, but it was only possible to examine the urine from three of these, as the other two died before satisfactory samples could be obtained. In each instance the urine was drawn off by catheter, shaken with a few drops of chloroform to preserve it, sealed in a glass vessel marked with a distinguishing letter and number, and at once despatched to London.

DOG A—ACUTE PANCREATITIS.

In the first dog (A) acute pancreatitis was set up by injecting turpentine into the main pancreatic duct (Plate, fig. 1), but the animal succumbed before a specimen of urine could be obtained for analysis.

Small bitch of the crossed dachshund type.

October 2, 1908: Scopolamine morphine (20 m) and ether narcosis. Incision through right rectus, 4 in. in length, and another just below the costal margin. Duodenum pulled into wound and incised opposite mesentery, about 3 in. from pylorus. Wirsung's duct readily found and sounded with probe. About 1 c.c. scopolamine morphine solution accidentally introduced with a small syringe, but immediately expressed. Atropine, 2 m (1 per cent.) given as an antidote, then 2 c.c. to 3 c.c. turpentine injected into the duct with a fine syringe. Gut closed with Lembert's sutures. Operation lasted about one hour forty-five minutes.

¹ Hoppe-Seyler's *Zeitschr. f. Physiol. Chem.*, Strassb., 1902, xxxv, p. 111.

² "Diseases of Metabolism," edited by R. C. Cabot, 1906, p. 262.

October 3: Dog fairly well, took some water, but apparently not thirsty, although tongue dry. Found dead 2 p.m.

Post-mortem, 5.30 p.m.: Rigor mortis present. The peritoneum contained about $1\frac{1}{2}$ pints of blood-stained odourless fluid, which, on bacteriological examination, was found to contain a pure culture of staphylococci. Omentum injected and slightly adherent in region of pancreas and over gut sutures. Incision into intestine apparently faultless and quite water-tight. Some spots of fat necrosis in omentum. A few hæmorrhages in the stomach wall and intense injection of all neighbouring intestine, especially descending colon. Pancreas swollen, dark red; tore in half in removing from abdomen. Wirsung's duct deeply injected. On cutting into the pancreas strong smell of turpentine. Bladder empty.

DOG B—SUBACUTE PANCREATITIS.

As the quantity of turpentine used for injection into the duct of Wirsung in dog A appeared to have been too much, a smaller amount was tried with dog B. It was proposed to employ 1 c.c., but, as the greater part of this escaped by the side of the needle or returned into the barrel of the syringe, it is probable that a much smaller quantity found its way into the duct. It was believed, however, that sufficient had been injected to set up some inflammatory changes in the gland.

Small black setter.

October 14, 1908: Urine drawn off by catheter (B^1). Abdomen opened in the middle line and duct of Wirsung found. Turpentine (1 c.c.) injected into the duct with a small syringe, but a good deal escaped into the lumen of the duct by the side of the needle, and, on releasing the pressure of the piston, much of the remainder reappeared in the barrel of the syringe, mixed with flocculent particles of (?) pancreatic juice. The wound was then closed.

October 15: Dog appears well; urine withdrawn by catheter (B^2).

October 16: Apparently well; urine withdrawn by catheter (B^3).

October 17: Still quite well, good appetite.

October 19: No apparent alteration having taken place, it was resolved to repeat the injection of turpentine. On opening the abdomen the omentum was found to be adherent to the sutures in the intestine and partly covering the pancreas, which appeared normal. A small piece from the edge of the gland was excised for microscopical examination. It was found that the nuclei of the cells were indistinct and the protoplasm of the cells much vacuolated, but there was no small, round-

celled infiltration or hæmorrhages such as are found in acute hæmorrhagic pancreatitis. The intestine was opened and a duct, which at the time was thought to be the duct of Wirsung, was found and injected with about $1\frac{1}{2}$ c.c. of turpentine. The intestine and abdomen were then closed. Urine withdrawn six hours after the operation (B^4).

October 20: A good deal of bilious vomiting. Urine withdrawn by catheter eighteen hours after operation (B^5).

October 21: Animal obviously suffering pain, so killed with chloroform. Post-mortem, pancreas normal to all appearance; gall-bladder adherent and contained ulcers; common bile-duct inflamed and necrotic. The turpentine had been injected into the *common bile-duct* and not into the duct of Wirsung, as had been intended. Residual urine in bladder withdrawn forty-eight hours after operation (B^6).

Examination of Specimen of Urine from Dog B.

Specimen B^1 , withdrawn before operation, received October 20, 1908: Reaction, alkaline; specific gravity, 1.052; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, negative.

Specimen B^2 , withdrawn sixteen hours after operation, received October 20, 1908: Reaction, alkaline; specific gravity, 1.039; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, both negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*. Pancreatic reaction: After standing for twenty-four hours the whole bulk of the fluid was filled with a light yellow flocculent crystalline precipitate, which, on microscopical examination, was found to consist of long, fine yellow crystals, which dissolved in 33 per cent. sulphuric acid in five to ten seconds (Plate, fig. 2). The melting point of the purified crystals was found to be 178° C. to 180° C.

Specimen B^3 , withdrawn twenty-four hours after operation, received October 20, 1909: Reaction, alkaline; specific gravity, 1.038; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*. Pancreatic reaction: In twenty-four hours a well-marked precipitate, but less than with Specimen 2; microscopically the precipitate was found to consist of typical sheaves of fine crystals, soluble in 33 per cent. sulphuric acid in five to ten seconds.

Specimen B^4 , withdrawn six hours after second operation, in which turpentine had been injected into the common bile-duct received

October 30, 1908: Reaction, faintly alkaline; specific gravity, 1.028; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, an exceedingly well marked green fluorescence with an alcoholic solution of zinc acetate. Pancreatic reaction, negative; no crystals found, even on microscopical examination, after twenty-four hours.

Specimen *B*⁵, withdrawn eighteen hours after second operation, received October 30, 1908: Reaction, acid; specific gravity, 1.026; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; urobilin, a slight green fluorescence with alcoholic zinc acetate. Pancreatic reaction, negative.

Specimen *B*⁶, urine withdrawn forty-eight hours after second operation, received October 30, 1908: Reaction, acid; specific gravity, 1.022; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*. Pancreatic reaction, negative.

DOG C—CHRONIC PANCREATITIS.

An attempt was made to induce chronic pancreatitis in this animal by providing a path by which intestinal bacteria might travel from the duodenum into the pancreatic duct, according to the method of Carnot.¹ A silk thread, about 5 in. long, was threaded into the duct of Wirsung from the intestine, 3 in. being introduced into the duct and fastened there; the remaining 2 in. were left hanging free in the duodenum.

Strong, active young female of the black-and-tan variety, weight about 30 lb.

September 18, 1908: Urine withdrawn by catheter (*C*¹). 2.30 p.m.: 0.05 grm. morphia injected subcutaneously. 2.40 p.m.: Vomited. 2.45 p.m.: Diarrhœa. 3.35 p.m.: Drowsy, but not comatose, abdomen prepared, ether narcosis. 4 p.m.: A 6 in. incision through the linea alba; all bleeding points secured. Peritoneum opened; stomach drawn into wound, but duodenum fixed and could not be drawn into the wound. Incision extended under left costal margin. The gut and pancreas then drawn up to wound, but, owing to fixation of duodenum, the common bile-duct could not be identified externally. A 4 in. incision was therefore made into the intestinal wall with the object of exposing the papilla of the duct; the gut was held above and below the incision by

¹ Gilbert et Thoinot, "Traité de médecine et de thérapeutique: Fasc. xx, Maladies des glandes salivaires et du pancréas," 1908, p. 238.

an assistant to prevent soiling of the peritoneum with intestinal contents. The papilla could not be found, even on compressing the gall-bladder. The lower part of the incision was therefore closed with fine silk, and the opening above extended so that the entire length of the incision was about 10 in. The pancreatic papilla was now seen, and a fine probe introduced. A silk thread (No. 3) was attached. The probe was easily felt through the gland substance, and blunt forceps were used to dislocate the gland lobules from the surrounding part of the duct wall, about 3 in. from the duodenum; scarcely any bleeding occurred. The end of the probe was then projected against the duct wall and the duct opened with scissors; the thread was then pulled through into the wound, threaded on a needle, and sewn to the surrounding trabeculæ. A small piece of omentum was sewn over the dislodged gland substance and the wound closed. The loose end of the thread in the duodenum was left about 2 in. long; the intestinal wall was sutured and the wound closed. The animal was in good condition when the operation was completed at 5.45 p.m. 11.30 p.m.: Animal lively and in good condition.

September 19, 9.30 a.m.: Dog very lively, sick once after drinking too much water; takes milk well.

September 20: Still lively and well; takes milk but refused small piece of rusk. Nose cold and wet; wound-covering keeps on well.

September 21: Dressing came off in afternoon; re-dressed. Fed 11 a.m. and 2 p.m. with raw meat; vomited second meal shortly after in large lumps. Right side of abdomen round stitches appears puffy; bowels not opened as yet. Urine drawn by catheter (C^2).

September 23: Dressing came off again; pus observed about upper angle of wound; wound re-dressed. Vomited. Normal motion.

September 25: Puppy born; no milk. All stitches septic and removed; sinus at upper angle of wound, but general condition good. No fresh dressing applied. Urine drawn off (C^3).

September 28: Appetite good. Sinus at upper end of wound open and discharging pus.

October 1: Stools very light colour, greasy, and offensive. Sinus not now discharging; wound almost healed. Gain in weight.

October 2: Stools not so light, but still very offensive. Urine drawn off (C^4).

October 6: General condition good.

October 9: Well. Urine withdrawn by catheter (C^5).

October 11: Well.

October 15: Extirpation of the pancreas. Incision to median side of old wound; pancreas exposed. In the region of the head of the gland a hard lump was felt. There were easily-broken-down adhesions between the horizontal part and the mesentery of the large intestine. The site of the sutures in the duodenum was covered by adherent omentum, but there was nothing to suggest stenosis of the gut. The pancreas was cleared of adhesions as far as possible, but there was considerable oozing from the gland substance. The vertical portion was then ligatured off from the mesentery on the one side and the gut on the other. As the gland was intimately amalgamated with the duodenum for about 3 in., it could not be dissected away, and some 6 in. of the duodenum was removed with the pancreas, and the cut ends joined with a Murphy button. There was a good deal of arterial hæmorrhage, but this was controlled by ligatures, and the wound closed by through-and-through stitches. Bladder emptied by catheter immediately the operation was completed. The excised gut, on being opened, was found to be thickened and narrowed. The silk thread was still in position, although much thickened and sodden. The whole pancreas, especially the head region, was thickened and felt heavier than normal. Microscopical examination of the gland showed marked overgrowth of the connective tissue in the neighbourhood of the duct of Wirsung and adjacent larger tributaries (Plate, figs. 3, 4). The epithelium of the ducts was loose and detached, in many places lying free in the lumen of the duct. In the neighbourhood of the smaller peripheral ducts there was some small, round-celled infiltration, and round cells were also seen in the walls and lumen of the ducts. The interlobular connective tissue did not show any marked change toward the periphery of the gland.

October 16: In the morning the animal had quite recovered from the operation. Very thirsty; frequent urination. Urine withdrawn sixteen hours after the operation, very offensive (C^6). About 4 p.m. (twenty-four hours after the operation) vomiting set in; heart-beats uncountable; little or no urine secreted.

October 17: Better. Three attempts to draw urine failed.

October 18: Found dead. Post mortem a fistulous opening, in the line of the skin incision, from which offensive intestinal gas escaped. Matted omentum and intestine underlying the abdominal wall shut in an abscess cavity in which lay the ends of the duodenum, all but separated, and between them lay the Murphy button disengaged, owing to ulceration through the partly necrotic walls of the intestine of the Lembert stitches covering the anastomosis. The stomach, intestine, &c.,

all showed signs of peritonitis, but there was no free fluid in the peritoneal cavity. No trace of pancreatic tissue found.

Examination of Specimens of Urine from Dog C.

Specimen C^1 , withdrawn September 19 (before operation), received September 29, 1908: Reaction, acid; specific gravity, 1024; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, negative.

Specimen C^2 , withdrawn September 21 (three days after operation), received September 29: Reaction, alkaline; specific gravity, 1028; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, crowds of fine long crystals, soluble in 33 per cent. sulphuric acid in five to ten seconds (Plate, fig. 5).

Specimen C^3 , withdrawn September 25 (one week after operation), received September 29: Reaction, acid; specific gravity, 1016; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, many long, fine crystals, soluble in 33 per cent. sulphuric acid in five to ten seconds.

Specimen C^4 , withdrawn October 2 (two weeks after operation), received October 13: Reaction, alkaline; specific gravity, 1032; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, some rather small, fine yellow crystals, soluble in five to ten seconds.

Specimen C^5 , withdrawn October 9 (three weeks after operation), received October 13: Reaction, acid; specific gravity, 1022; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, some typical long, fine crystals, soluble in 33 per cent. sulphuric acid in five to ten seconds.

Specimen C^6 , withdrawn October 18 (fifteen hours after extirpation of the pancreas, the bladder having been emptied on completion of operation), received October 20: Reaction, alkaline; specific gravity, 1046; albumin, *nil*; sugar, Fehling, instant reduction; phenylhydrazin, crowds of coarse crystals in sheaves, insoluble in 33 per cent. sulphuric acid in five minutes (Plate, fig. 6); melting point, 204° to 205° C.; quantitatively, Bang's method 1.8 per cent., fermentation (Lohenstein)

1.6 per cent., polariscope + 1.2 per cent.; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*. Pancreatic reaction: The urine was filtered, and 45 c.c. of the filtrate made faintly acid with HCl; 3 c.c. of HCl were then added, and the mixture boiled for ten minutes. The excess of acid was neutralized with lead carbonate and the glycuronic acid removed from the acid filtrate by shaking with tribasic lead acetate. The lead in the filtrate was then precipitated with a stream of sulphuretted hydrogen, and removed by filtration. After being boiled for a few minutes to expel the excess of sulphuretted hydrogen, the filtrate was cooled and mixed with half its bulk of distilled water. Yeast was then added, and the mixture incubated at 37° C. for eighteen hours, when, as it was found that a control specimen no longer gave any reaction for sugar, it was filtered, and 15 c.c. of the filtrate mixed with 3 c.c. of water, 0.75 grm. of phenylhydrazin hydrochlorate, and 2 grm. of sodium acetate. The mixture was boiled for ten minutes and filtered twice, while hot, through a hot, moist filter-paper. Examination twenty-four hours later showed no crystalline deposit, either to the naked eye or microscopically.

DOG D—EXTIRPATION OF THE PANCREAS.

Female mongrel, aged about 3 years and weighing about 10 lb., recently in pup and still lactating.

September 1, 1908, 4.45 p.m.: Given $\frac{1}{3}$ gr. morphia subcutaneously; copious salivation in about five minutes, drowsy a few minutes later, but not completely unconscious, pupils medium size. Abdomen shaved and sterilized, limbs and chest wrapped in cotton wool; ether narcosis. Incision in middle line from sternum to umbilicus; peritoneum opened and held with stitches; pancreas found, tail easily isolated without much hæmorrhage; horizontal part markedly developed; vessels large and needed much care in tying, but were eventually successfully separated; just as the head was being detached through an additional horizontal incision a large vessel was cut and was only found and ligatured with difficulty, as it retracted into the peritoneum; a large amount of blood was consequently lost. The body wall was hastily sewn up, all the layers together, an infusion of 100 c.c. of saline given into the back, and the animal placed between hot bottles on a paraffin cupboard at 6.30 p.m. At 11 p.m. animal still alive and condition improved.

September 2, 7.30 a.m.: Dog found dead; rigor mortis present, but body still warm. Immediate autopsy revealed a somewhat congested

duodenum, some blood-clot in the pelvis, no sign of peritonitis, no obvious source of hæmorrhage, bladder empty, pancreas apparently completely removed.

DOG *E*—EXTIRPATION OF THE PANCREAS.

The pancreas of this animal was extirpated on September 9. On September 11 it died with gangrene of the duodenum. Post mortem, no trace of pancreatic tissue could be found. A specimen of urine was obtained for analysis on September 10.

Female fox-terrier mongrel, weight 18 lb. to 20 lb.

September 7, 1908: Urine drawn off by catheter under ether narcosis (*E*¹).

September 9, 3.10 p.m.: Morphia ($\frac{1}{3}$ gr.) subcutaneously. 3.15 p.m.: Vomited. 3.30 p.m.: Diarrhœa; abdomen shaved and sterilized, limbs and chest wrapped in cotton wool; light ether narcosis. 4.15 p.m.: S-shaped incision into the abdomen, all bleeding stopped; peritoneum opened, cut edges stitched to sterile gauze apron; head of pancreas found in duodenal loop, from which it was carefully separated; a good deal of hæmorrhage, controlled with difficulty; the body and tail then dissected out. 6.15 p.m.: Extirpation of the pancreas complete and abdominal wall stitched up in two layers; finished. 6.35 p.m.: Dog cold and no pulse at wrist; gauze, leukoplastic and collodion dressing applied. 9 p.m.: Heart-beats 148, no pulse at wrist. 11 p.m.: Drank water.

September 10, 8 a.m.: Thirsty, but otherwise well. 9 a.m.: Vomited. 12 a.m.: A large quantity of urine drawn off by catheter (*E*²). 10 p.m.: Well; walks when shown a basin of water, but does not lap very greedily; refuses milk absolutely.

September 11, 6 a.m.: Dog standing at door; lapped some water, but not greedily. 7 a.m.: Not so lively. 10 a.m.: Very listless and obviously ill; abdomen not markedly distended or tender, respirations a little increased, heart-beats rather faint, ? dullness right flank. 12 a.m.: Weaker; respirations peculiar—thoracic inspiration associated with indrawing of abdomen, a pause, then abdomen somewhat suddenly protruded. 3.15 p.m.: After a few convulsive respiratory efforts the animal died. Immediate post-mortem: Stitches healed well, no inflammatory reaction; on opening the peritoneum about an ounce of turbid, blood-stained fluid was evacuated from an abscess cavity about the duodenum, under the great omentum; the duodenum itself, just where

the bile papilla is situated, was the seat of a circular patch of gangrene in which were two minute perforations, from which gas exhaled; recent adhesions everywhere; gall-bladder contained bile under pressure through blocking of the duct at the papilla: two small spots of fat necrosis; no pancreatic tissue found.

Examinations of Specimens of Urine from Dog E.

Specimen *E*¹, withdrawn September 9 (before operation), received September 19: Reaction, alkaline; specific gravity, 1042; albumin, *nil*; sugar, Fehling and phenylhydrazin tests, negative; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, *nil*; pancreatic reaction, negative.

Specimen *E*², withdrawn September 10 (sixteen hours after operation), received September 19: Reaction, acid; specific gravity, 1040; albumin, *nil*; sugar, Fehling, instantly reduced; phenylhydrazin, crowds of sheaves of coarse crystals, insoluble in 33 per cent. sulphuric acid in five minutes; melting point of the purified product, 204° to 205° C.; quantitatively, Bang's method 3.9 per cent., fermentation (Lohenstein) 3.8 per cent., polariscope + 3.2; acetone, *nil*; aceto-acetic acid, *nil*; bile, *nil*; urobilin, a slight green fluorescence with an alcoholic solution of zinc acetate; pancreatic reaction (carried out as in *C*⁶), no crystalline deposit, either macroscopically or microscopically, in twenty-four hours.

The results of these experiments confirm the conclusions previously arrived at from clinical observations and post-mortem examinations in the human subject. They show that while normal urines give no reaction with the improved, or C— "pancreatic" reaction, specimens derived from animals in which inflammatory changes have been set up in the pancreas give a more or less well-marked reaction according to the extent and intensity of the degenerative changes in the gland. The analyses of the specimens from dog *B* are particularly instructive, for, although only a very small quantity of turpentine was injected into the pancreatic duct, since a larger amount had rapidly produced a fatal hæmorrhagic pancreatitis in dog *A*, it was sufficient to cause a well-marked pancreatic reaction in the urine withdrawn sixteen hours later. A second specimen, taken twenty-four hours after the operation, gave a less marked, but still distinct, reaction. A third sample obtained five days after the first operation and six hours after the second, in which 1.5 c.c. of turpentine was injected by mistake into the common bile-duct, was, however, negative, as also were two others

obtained twelve and forty-two hours later, thus showing that the turpentine itself was not responsible for the previous positive results, but that the urinary changes were dependent upon the effects produced on the pancreas. As only a very small quantity of turpentine was injected into the pancreatic duct at the first operation and none at the second, it is probable that the injury of the gland was not very widespread or severe, so that the negative result of the pancreatic reaction in the specimen of urine obtained on the fifth day after the operation, and the absence of any noteworthy changes in the gland when it was examined a week after the operation, are not surprising. An interesting effect of the accidental injection of turpentine into the common bile-duct of this animal at the second operation was the appearance of a very well marked reaction for urobilin in the urine, for, from clinical experience, we have come to regard such a reaction as an indication of cholangitis, and have noticed its very constant association with the presence of floating gall-stones in the common bile-duct, whether these are accompanied by jaundice or not.

Carnot has stated that chronic pancreatitis may be produced in dogs by establishing a route by which the bacterial contents of the duodenum may travel up into the pancreatic ducts, and for this purpose he passes a silk thread from the intestine along the main duct. Our experience with dog *C* confirms this observation. The silk thread introduced at the operation was still in position when the parts were examined twenty-eight days later. The whole pancreas was then found to be heavier and denser than normal. Microscopical examination showed a fibrosis affecting the interlobular strands, particularly in the neighbourhood of the ducts. The epithelium of the larger ducts was desquamating and the walls of the smaller ducts showed evidences of catarrhal changes with some small round-celled infiltration. The urine withdrawn three days after the operation, when it was probable that the inflammatory reaction in the pancreas was well established, gave a very characteristic "pancreatic" reaction. One week later the reaction was somewhat less marked. Two weeks after the operation a positive result was still obtained. A specimen withdrawn three weeks after the thread had been introduced also gave a positive reaction, but the crystals were much less numerous than in the first sample. After extirpation of the pancreas, although the urine contained nearly 2 per cent. of sugar, no "pancreatic" reaction could be obtained. It was thus apparent that the chronic inflammatory changes induced in the pancreas by the introduction of a silk thread connecting the duct of Wirsung with the

duodenum were associated with the appearance in the urine of an intense "pancreatic" reaction, which gradually became less marked with the lapse of time, but still persisted three weeks after the performance of the operation. Although it is probable, as Carnot suggests, that the passage of bacteria from the intestine into the pancreatic duct is an important factor in producing the resulting pancreatitis, we are inclined to think that the partial obstruction of the duct by the thread, and consequent stagnation and back pressure of the pancreatic secretion, play some part in the process. The more marked microscopical changes in the neighbourhood of the larger ducts and the gradual subsidence of the "pancreatic" reaction suggest limitation of an infective inflammatory process, originating in the ducts, by a barrier of connective tissue, with lessening destructive changes in the gland substance, but accommodation of the pancreas to the changed pressure conditions had possibly also some influence on the course of the urinary changes.

Total extirpation of the pancreas is a difficult operation to carry out successfully, and the first animal, *D*, in which this was attempted, died thirteen hours after the operation, before a specimen of urine had been obtained for analysis. The second dog lived two days. In this animal, *E*, a specimen of urine withdrawn eighteen hours after the operation contained 3.9 per cent. of sugar, but gave no "pancreatic" reaction, suggesting that the reaction does not depend upon disturbances of metabolism due to loss of the internal functions of the pancreas, but arises from tissue changes in the gland itself. Our experience with dog *C* points still more clearly to this being the explanation of the reaction, for in this animal, although four previous specimens obtained at intervals during the preceding three weeks (the last, one week before the pancreas was removed) had given a characteristic positive reaction, a sample withdrawn fifteen hours after the operation gave no reaction whatever, after the 1.8 per cent. of sugar present had been fermented off, indicating that the extirpation of the pancreas had removed the source from which the mother-substance of the crystals obtained in the previous positive reactions was derived. It may be objected that the modification of the test made necessary by the presence of a fermentable sugar interferes with the reliability of the test, but the experience of one of us with several cases of undoubted pancreatic diabetes in the human subject has shown that a positive "pancreatic" reaction is obtained by this method whenever there is reason to think that active degeneration of pancreatic tissue is going on.¹

¹ *Surg., Gyn., and Obstet.*, Chicago, 1908, vi, p. 22; *Proc. Roy. Soc.*, 1909, B, lxxxi, p. 375.

EXPLANATION OF PLATE.

FIG. 1, DOG A.—Acute hæmorrhagic pancreatitis following the injection of 2—3 c.c. of turpentine into the main pancreatic duct (\times ca. 50).

FIG. 2, DOG B.—Crystals obtained from the urine by the C. pancreatic reaction sixteen hours after the injection of less than 1 c.c. of turpentine into the main pancreatic duct (\times ca. 200).

FIG. 3, DOG C.—Section through the duct of Wirsung (\times ca. 50).

FIG. 4, DOG C.—Section in the neighbourhood of a main tributary of the pancreatic duct (\times ca. 50).

FIG. 5, DOG C.—Crystals obtained from the urine by the C. pancreatic reaction three days after Carnot's operation (\times ca. 200).

FIG. 6, DOG C.—Glucosazone crystals obtained from the urine after extirpation of the pancreas (\times ca. 50).

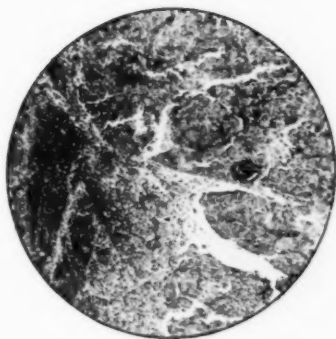


FIG. 1.

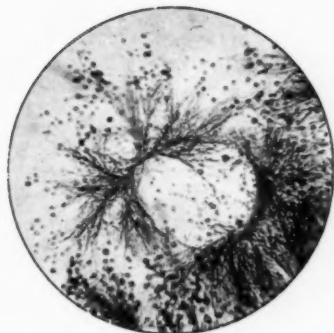


FIG. 2.



FIG. 3.

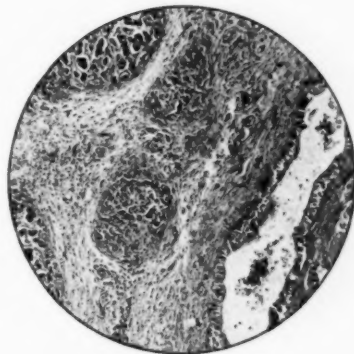


FIG. 4.

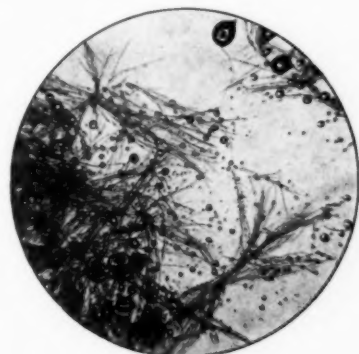


FIG. 5.



FIG. 6.



A Case of extensive yet incomplete Fibro-caseous Disease of both Suprarenal Capsules in which Symptoms of Addison's Disease were not present.

By HERBERT FRENCH.

G. B., AGED 48, a painter by trade, came under observation on account of a lump on the inner side of his left knee; this lump had first appeared a year previously, and it had greatly increased in size so as to interfere with walking. There was now a large swelling over the internal condyle of the left femur and upper part of the tibia. The diagnosis at first lay between myeloid sarcoma on the one hand and tuberculosis of the knee-joint upon the other. The trouble was ultimately shown to be tuberculous arthritis, upon which acute suppurative infection subsequently developed and caused the patient's death. At the post-mortem examination, in addition to the local mischief in the knee and leg, extensive adhesions were found all over both lungs, and, in addition to scarring of the apices of both upper lobes, there was a fibro-caseous focus as big as a bean near the apex of the left upper lobe, and dense fibrous adhesions without caseous material at the apex of the right upper lobe. There had obviously been extensive phthisis in both upper lobes, but this had got perfectly well, and there was now no active tuberculosis of the lung. The only other organs that merit detailed description are the suprarenal glands. Each of these was of about normal size and shape, and, before being cut into, the only point that attracted one's attention in them was their firmness, which was comparable to the firmness of the suprarenal glands in cases of chronic heart failure. When each was cut into, however, it was found that there was considerable fibro-caseous degeneration in both of them. There was, however, a sufficiency of suprarenal tissue still left, representing about one-quarter of the right suprarenal capsule, and about one third of the left. The line of distinction between normal gland substance and the firm fibro-caseous tissue was very sharp, so that the process had presumably been going on for a long time. Nevertheless the section here exhibited shows that there are numerous giant-cell systems in the peripheral part of the caseous portion of the gland, so that presumably the disease was not obsolete, but slowly progressing. About half of the section is

structureless and caseous, half is normal, the two parts merging into one another without any structural line of demarcation between them. The oldest caseous change seems to be central, the giant-cells indicative of more recent tuberculous infection being round the edge.

The interest of this case lies mainly in the fact that it affords to some extent a natural experiment upon how much less than the normal amount of suprarenal gland tissue a full-grown human being may have without developing symptoms of Addison's disease; in the above case the amount of suprarenal tissue left amounted to a little over one-quarter of the normal total.

**Bronzed Diabetes, with Cirrhosis of both Liver and Pancreas;
and Pigmentary Deposits in these Organs as well as in
the Skin.**

By HERBERT FRENCH.

C. S., AGED 55, came under observation suffering from glycosuria, giving a history that, although he had been a heavy drinker, he had been quite well until eight months previously, when he noticed that he was wasting, and shortly after that he developed an abnormal thirst. Sugar was found in the urine and a strict diet was adopted, but without benefit. The urine contained sugar, albumin without tube casts, acetone, and diacetic acid. He was a tall man, but exceedingly thin, weighing little over 8 st., though his height was 5 ft. 10 in. There was extensive pigmentation of the skin. In addition to the general bronzing of the body there were many very dark pigmentary deposits on the legs between the knees and ankles, and also a large number of freckle-like spots on other parts of the body. There was no similar pigmentation of the buccal mucous membrane. The degree of pigmentation was less than has been observed in some other cases of bronzed diabetes, but there was no doubt as to the diagnosis in this case.

The heart and lungs were natural. It is noteworthy that the aorta was remarkably healthy and free from atheroma, notwithstanding the patient's habits. The only organs that merit special attention are the kidneys, spleen, pancreas, liver, and the intestines. The intestines throughout their length exhibited upon their serous surface a characteristic broad white line on either side of the dark-red blood-vessels; this is an appearance which is fairly common in diabetic cases even when

there is no lipæmia—and there was no lipæmia in this instance. The white lines appear to be lymphatics distended with some fatty substance. The spleen weighed 346 grm., being enlarged and firm, as in most cases of cirrhosis of the liver. The kidneys together weighed 538 grm. They were symmetrical organs, and looked natural to the naked eye except for their great bulk. Their increased size was due no doubt partly to the diabetes, but partly also to the patient's former habits of drinking. The capsules peeled well, leaving a perfectly smooth surface, free from cysts, and the renal arteries looked natural. There was no microscopical evidence of nephritis to account for the albuminuria, and the microscopical section showed that there was no interstitial fibrosis, and that the epithelial cells exhibited no change that might not have been entirely due to post-mortem degeneration. There is no obvious pigmentation in the renal cells. The liver weighed 2,551 grm. It was a big pale brown, tough, cirrhotic liver of the characteristic small hobnail type. The section shows an extreme degree of peribulbar fibrosis with proliferated bile-ducts in the fibrous tissue, and also, what is worthy of special notice, a very marked degree of dark-brown pigmentary deposit, particularly within the epithelial cells many of which also show large fat droplets, and in the cells of the so-called proliferated bile-ducts. Hardly a cell is free from the pigment, which is thickly peppered all through them; there is little, if any, extra-cellular pigment. The pancreas weighed exactly 100 grm., and, although it is often difficult to demonstrate macroscopical changes in this organ in cases of diabetes, in this case it exhibited very obvious abnormality. It was exceedingly firm, and one could readily see broad bands of fibrous tissue and fat infiltrated between the lobules of the gland, the lobules themselves being thereby considerably reduced in size; a great deal of the weight, therefore, was due not to pancreatic but to fibrous tissue, and the condition might well be described as cirrhosis of the pancreas comparable to the cirrhosis of the liver. The colour of the parenchymatous tissue of the pancreas was of a remarkable and peculiar rusty brown, and the microscopical section exhibits atrophy of many of its alveoli, together with considerable increase of the interstitial tissue, both fibrous and fatty. Some of the gland cells look normal, but in some parts the parenchyma is less easily recognisable, and in many of the cells in these places there is an abundant deposit of brown pigment granules precisely similar to those seen in the hepatic cells. The arterioles do not seem to be unduly thick-walled; the islands of Langerhans are still to be detected in some places, but many of the cells in

them show brown pigment granules like those in some of the alveolar cells.

The pigmentary changes in the pancreas and liver are presumably related to the pigmentary changes in the skin. They have been described by many previous observers, especially in France by V. Hanot,¹ A. Marie,² M. E. Jeanselme,³ de Massary and Potier,⁴ and others.

My only excuse for bringing the specimens before you is that they are not to be met with very commonly. The pigment granules are ferruginous, and I have here sections both of the liver and of the pancreas that have not been stained, but have simply been treated, as in Perl's test, with potassium ferrocyanide and hydrochloric acid; they have been turned a bright Prussian-blue colour, and, under the microscope, it is clearly seen that each intracellular pigment granule has been turned a bright-blue colour by this simple chemical test for iron. The pigment is therefore presumably derived in some way from blood pigment, though possibly only in an indirect manner. It does not seem to be related to any particular changes in the suprarenal capsules; the granules have been found in a great many different organs, so that the pigmentary changes are not local but widespread. It is not yet settled, however, whether the granules are merely taken up from the blood by the cells and, so to speak, stored, or whether they are produced within the cells themselves. Some observers have reported abundance of extracellular pigmentary deposits, but in the present instance its intracellular localization is a striking feature; moreover, the healthier looking the cells are, the fewer the pigment granules they contain, the pigmentary deposits seeming to be directly associated with other signs of cell-degeneration, such as alteration in shape, diminution in size, and diminished capacity for nuclear staining, together with the accumulation of intracellular fat droplets, particularly in the liver.

In some cases one can see the blood-vessels distended with red corpuscles, and no pigment granules are to be seen within these vessels. One's general impression is, therefore, that the pigment granules have been produced within the cells themselves.

¹ "Diabète Bronzé," *Brit. Med. Journ.*, 1896, i, pp. 206-7.

² "Sur un cas de diabète bronzé suivi d'autopsie," *Semaine Méd.*, 1895, xv, p. 229.

³ "Pathogenie du diabète bronzé," *Bull. et Mem. de la Soc. méd. des Hôp. de Paris*, 1897, xiv, sér. 3, pp. 179-203.

⁴ "Un cas de diabète bronzé," *Bull. de la Soc. Anat.*, 1895, sér. 5, ix, pp. 354-362.

A Chylous Cyst Excised from the Neck.

By E. O. THURSTON.

R. P., HINDU, male, aged 30, was admitted in the Gaya Pilgrim Hospital on September 20, 1909, with a cyst of the neck. Two years before admission he had a few days' fever, about which nothing characteristic could be elicited; it is very possible that it was malarial in nature. Shortly afterwards a swelling appeared just above the left clavicle, which disappeared under the application of *tinctura iodi* after a week or so. A year later the tumour again made its appearance, and since then there has been a slow increase. There was no more fever, and various irritants had been applied without effect. The patient had noticed a periodic increase in the size of the swelling, two or three hours after eating; he had the usual two meals a day—at 10 a.m. and 9 p.m. It was always distinctly smaller in the morning.

On admission he was a well-nourished man. There was a swelling rather smaller than a tennis ball, slightly oval in the transverse direction, and situated just above the left clavicle. There were marks of old counter-irritation over the cyst, which was not adherent to the skin. Fluctuation was present and the cyst was freely movable; it extended beneath the sterno-mastoid, and could be pushed further that way; there was no pain.

On September 22 the cyst was exposed by a transverse incision; on opening it there was a gush of typical chylous fluid with a distinct tinge of red. A finger was introduced into the cyst, and it was very easily dissected out. As more chyle was coming from near the position of the entrance of the thoracic duct into the vein, the position of the cyst on this side was not dissected out, and a pedicle was made of it, which was ligatured with silk, the remainder of the cyst being cut away. After the cyst had been removed several dilated lymphatics were seen on the floor of the subclavian triangle. The largest, which was about the size of string used for tying up small parcels, and filled with chyle, ran from about the level of the junction of the upper $\frac{1}{3}$ with the lower $\frac{2}{3}$ of the jugular vein, downwards and outwards to the subclavian vein; a side branch of the latter had been cut, and this was ligatured. Not wishing to damage any anastomotic circulation, I refrained from tracing out the course of the lymphatic vessel referred to further. The cyst on its inner surface was granular and finely trabeculated, and presented larger circular

areas, each about the size of a threepenny-piece, at the centre of which there was a knot with fine fibres radiating to the surrounding trabeculæ, the appearance recalling that of the veining of a leaf.

After a few days chyle commenced to discharge through a small portion of the wound at its outer extremity, and persisted without diminution until October 13, three weeks after the operation. The amount discharged was very considerable—several fluid ounces; roughly speaking, 4 oz. would be about the average. No correct estimation was practicable, but fairly voluminous dressings were quickly soaked, and, after eating, chyle would run down underneath these over the patient's chest.

On October 13 the cavity left, which was still large, was tightly plugged with gauze, and there was no further soaking of the dressings. The plug was removed on October 18, and after its removal no further escape of chyle took place. Gauze was again put into the wound and removed on October 22; the pressure caused the wound to open out, but it was completely healed by November. The wound ran an aseptic course, although there were many opportunities for its infection.

The chyle had the same faint red tint throughout, and its loss did not interfere with the nutrition of the patient, who, in fact, appeared somewhat fatter on his discharge from the hospital than when he was admitted.

A Large Chylous Cyst of the Mesentery.

By S. G. SHATTOCK.

THE opportunity of examining a chylous cyst is so rare that any example which comes under notice is worth placing on record, and I may add the following to Mr. Thurston's case. The cyst itself must be put down as the indirect cause of death, from its mechanically having led to volvulus of the intestine. The patient was a man, aged 22, who was admitted to the Tiverton Infirmary, Devon, with the following history, which was kindly supplied by Mr. P. J. de Miranda: He was suddenly taken ill on January 17, 1910, with a very acute pain in the lower abdomen; he became faint and vomited twice; the pain subsided, but persisted till death, which occurred a week later. He micturated naturally twice after the onset of his illness, but as the bowels were constipated he took a purgative on the third day, which acted. He then found himself unable to urinate. A catheter was passed with difficulty,

but only a few ounces of urine were withdrawn. When subsequently admitted to the Infirmary, Mr. Miranda noticed a rounded swelling occupying the hypogastric with part of the umbilical, the right iliac and lumbar regions; this was dull on percussion, not tender, and altogether like a distended bladder.

As micturition was impossible, a No. 3 catheter was passed, though with much difficulty, the instrument being gripped about the prostatic region as if in a stricture; six ounces of urine were withdrawn. The urine was dark, blood-stained, not offensive, and contained no pus. On examination, *per rectum*, there was felt the posterior part of a sac of fluid fully occupying the pelvic cavity and kinking the rectum, so as to make it difficult to introduce an enema tube. The bowels acted daily, and the evacuations were normal. The catheter was used regularly. He improved till January 23, when the pain returned worse than it had been before; he became blanched and cold, and died on January 24. After death a large cyst was found impacted in the pelvic cavity. The bladder was tightly pressed against the pubes and empty. The cyst and portion of small intestine above it were dark red, and the latter distended, as if from a twist above the seat of the cyst; the small gut below and the colon were of natural colour. The other organs presented nothing abnormal. The parts were removed and sent to the Royal College of Surgeons.

Description of the Specimen.—The cyst in question is spherical, about 12.5 cm. (5 in.) in diameter, and, for its size, thinly walled, being, when stripped of peritoneum, less than 2 mm. in thickness. It lies in the mesentery in contiguity with the small intestine, a long, intensely-congested coil of which almost surrounds it like a frill. The cyst is not fairly in the mid-substance of the mesentery, but projects chiefly from one aspect, which is immediately covered with separable though abnormally-adherent peritoneum, whilst the fat of the mesentery lies over it on the opposite, less prominent side. On its exterior there are patches of old, firmly-adherent, shagreened adhesions. Its interior is smooth and without any cutaneous areas.

On puncture I found it to be completely filled with a thin, creamy, slightly blood-stained fluid, without hair or other elements. It should be stated that no preservative had been used before the evacuation of the contents. Microscopically, the fluid contained no leucocytes or other cell forms, and on shaking a sample with ether, letting it stand for twenty-four hours, decanting the ether, and allowing the latter to evaporate, a well-marked residue of fat was left at the bottom of the vessel.

Action of Thyroid and Antithyroid Preparations.

By WALTER EDMUNDS.

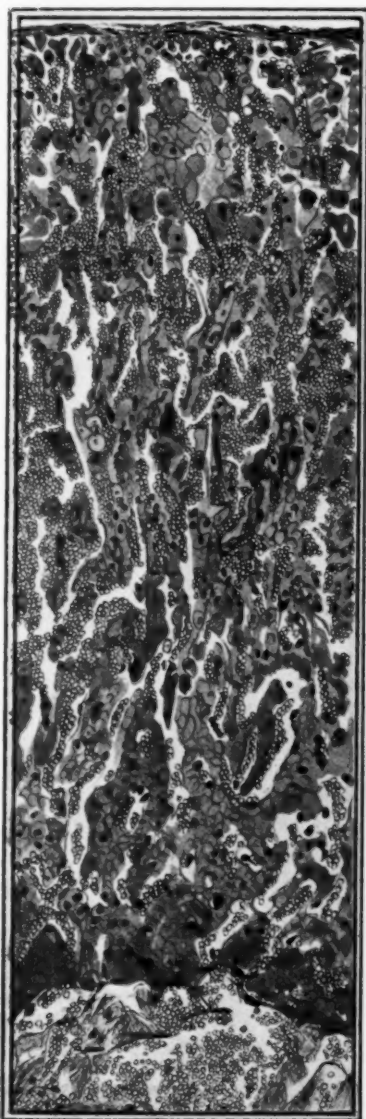
THE experiments were made on rats with commercial thyroid and antithyroid preparations. Altogether ten experiments were made and four controls used; the controls remained well throughout. The preparations were given by the mouth in doses of 10 gr. three times a day, mixed with food. One thyroid preparation produced on two rats death or severe illness in twenty days. Another thyroid preparation produced the same result on two other rats in eighteen and nineteen days. An antithyroid preparation produced no effect on two rats during thirty-nine days. Thyroid and antithyroid preparations administered together, 10 gr. of each three times a day, caused death or severe illness in ten, eighteen, nineteen, and twenty-one days. The symptoms produced were loss of flesh, roughness of coat, general weakness and illness, and death.

Post-mortem: The chief change found was marked alteration in the suprarenal glands; they were obviously enlarged and dark in colour as compared with the glands in the controls; under the microscope they were much engorged with blood and contained hæmorrhages (see fig.). The other viscera, including the thyroid glands, showed no marked or uniform changes.

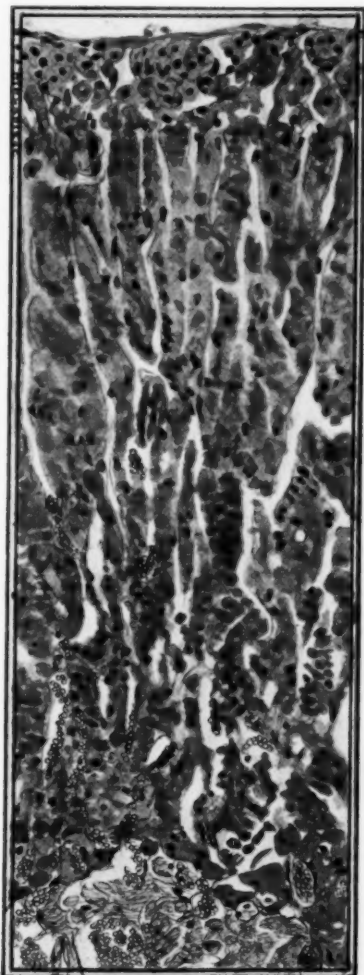
The conclusions drawn are: (1) That thyroid feeding in large doses causes congestion of, and hæmorrhage into, the suprarenal glands; (2) antithyroid preparations in equal doses with the thyroid given produces no result in warding off the ill-effects of thyroid excess.

REFERENCE.

- DUDGEON, LEONARD. "The Etiology, Pathology, and Diagnosis of Adrenal Hæmorrhage,"
Amer. Journ. Med. Sci., Philad., 1904, cxxvii, p. 134.



Cortex of suprarenal gland of rat fed with thyroid extract, showing congestion and hæmorrhage.



Cortex of suprarenal gland of normal rat.

The Influence of Oöphorectomy upon the Growth of the Pelvis.

By S. G. SHATTOCK and C. G. SELIGMANN.¹

WHEN we recall the widespread and diverse effects induced by early castration upon the growth of the accessory organs of generation—the prostate, the vesiculæ seminales, and Cowper's glands, not to mention the arrest or modification resulting in the secondary or external sexual characters—we are naturally led to ask how far removal of the ovaries in the young is followed by a corresponding hypoplasia or undergrowth in the accessory parts of the generative apparatus in the female. These parts comprise not only the uterus, vagina, and mammary glands, with the glands of Bartholin, but also the pelvis, and it is with the last-named that the present communication deals. The case as it concerns the uterus (in the rabbit) has been already dealt with by one of us (S. G. Shattock) in the *Proceedings of the Royal Society of Medicine*, December, 1909.² Our own observations upon the hypoplasia of the accessory organs which follows castration in the lamb are recorded elsewhere,³ and need not be, for the present purpose, again detailed.

In the case of the human subject similar results are observable after castration or complete ablation of the external genitalia, although the two of which we ourselves know are less satisfactory, for the reason that the precise age at which the mutilation had been carried out is not forthcoming, and, the subjects having lived for some years after puberty,

¹ The expenses incurred by these observations were defrayed partly by a grant from the Scientific Grants Committee of the British Medical Association, and partly by the Lister Institute, at the Elstree farm of which the calf to be referred to, was kept for the chief part of the time.

² In this paper the striking hypoplasia of the uteri resulting from oöphorectomy carried out upon the young rabbit is illustrated by figures showing the diminutive uteri in the spayed animal as compared with those of one that had been pregnant for the first and only time three months before being killed. To render the comparison more complete I may add the measurements of the uterus and tube of a fully-grown doe (12 months) which had never been "covered," and which weighed 2 kilos 950 grm. before being killed; the oöphorectomized doe having weighed 3 kilos 280 grm. Length of right uterus along its curves, 8 cm. Diameter of right uterus, 1 cm. Length of right Fallopian tube along its curves, 11 cm. Buys and Vandervelve have described the microscopic appearances of the uterus in the rabbit after oöphorectomy.

³ *Proc. Roy. Soc.*, lxxiii, and *Trans. Path. Soc.*, lvi, p. 57.

it is impossible to estimate how far the diminutive size of the accessory organs may be due to arrested growth and how far to secondary wasting.¹ In the Museum of the Royal College of Surgeons there is a specimen (No. 25, Pathological Series) showing part of the pelvic viscera of an adult Russian from whom the penis, scrotum, and testes were removed, probably in early adult life. The scrotum is represented by a flattened eminence of cicatricial tissue, and above it there is a similar eminence in which is the orifice of the mutilated urethra. The prostate is diminutive, the vasa deferentia are thread-like, and the vesiculæ seminales are without their natural pouchings. The parts were obtained from one of the castrating Russian sect known as Skoptzi ("the castrated"). A full account of the sect is given in a monograph by Dr. E. Pelikan.² In one set of cases the scrotum, with the testicles, is alone removed; in another, the penis is swept off at the same time. No particular age is enforced for the practice of the mutilation, which is carried out sometimes in the young, sometimes in the fully-grown adult.

We are able to confirm this observation by our own study of a specimen which has recently been presented to the College of Surgeons by Professor A. F. Dixon (Dublin), and which we may here describe. The parts comprise the generative organs and pelvis of a man who was for many years a ward-attendant in a Union hospital. He always kept to himself; his voice was peculiar, and was described as "rather squeaking"; he never shaved. He was born in China, his mother being Chinese and his father British. At the time of death he was an old man. The hair on his scalp was dark, thick, and soft; there were but few hairs on his face; the pubic hair was scanty. His height was 5 ft. 5½ in. A low, elliptical eminence marks the site of what remains of the free portion of the penis, and in the summit of a small papilla on this is the urinary meatus. The scrotum has been completely removed. The dissection of the parts shows that the deep remnants of the corpora cavernosa and corpus spongiosum are normally disposed, and that the low eminence representing the stump of the penis is due to the presence of a redundant portion of these bodies, which are bent upwards at an acute angle. The external meatus, which lies in the divided end of the corpus spongiosum, is a vertical slit 7 mm. in length. The size of these structures is neither that of childhood nor of the fully-grown adult.

¹ It is stated that if castration is carried out after puberty the beard diminishes but does not disappear. *Les Eunuques de Constantinople*, Drs. Hikmet et Regnault (loc. cit.).

² "Gerichtlich-medizinischen Untersuchungen über das Skopzenthum in Russland," Giessen, 1876.

The vasa deferentia, which are reduced to threads, preserve their normal anatomical relations, and above the prostate gland, on the outer side of each duct, there lies a diminutive vesicula seminalis. Each vesicula is 3 cm. in length, and is a slender, non-sacculated cord-like structure not more than 3 mm. in diameter; the diameter of the vas in the same situation is 2 mm. As regards the prostate gland, this measures no more than 1.5 cm. antero-posteriorly in the sagittal section; and 2 cm. in the extreme vertical direction. Traces of gland tissue appear to the naked eye in the presence of a few groups of minute cystic spaces.

Microscopic sagittal sections show that the chief part of the organ consists of closely-set bundles of unstriated muscle fibre united by a thin net of fibrous tissue. The amount of gland tissue is small; of this there are areas, however, which present quite a normal structure; cystic dilatations occur here and there. The urethral epithelium is normal.

The Influence of Double Oöphorectomy upon the Growth of the Pelvis.¹

That the growth of bone may be modified by the absence of certain internal secretions appears from some of the results of castration and of disease. After castration, carried out in the young of the Herdwick sheep, a variety in which the male alone is furnished with horns, not only is the growth of the horns (as we have found) completely prevented, but, in addition to this, the normal change of form which the male skull undergoes, no longer takes place. The hornless skull of the castrated Herdwick resembles that of the ewe; the plane of the os frontis is continued backwards behind the orbits at a very obtuse angle, whilst in the intact ram the plane of the frontal behind the orbit lies almost at a right angle with the inter-orbital portion of the bone, the horn-cores arising from the upper or horizontal area. One might be tempted at first to ascribe this failure of cranial change to the absence of horns, but more probably both results are due to the same cause, seeing that the horns of the sheep do not come, like those of the rhinoceros, into the category of mere cutaneous appendages, but are provided with osseous cores. In regard to the long bones, one of the well-recognized results of castration carried out in the young animal is an increase of their length. In the case of our own six Belgian hares (to be presently referred to)

¹ Certain of the functions of the ovary as an organ of internal secretion have been discussed by F. H. A. Marshall and W. A. Jolly (*Phil. Trans.*, Royal Society, 1905-6, p. 123). These deal with phenomena connected with pregnancy, and oestrus or "heat."

the lengths of the right femur and of the right humerus in the castrated male [markedly exceed the length of the corresponding bones in the normal male, in two normal females, and in two spayed females. Taking the two males as fairly adapted for comparison :—

CASTRATED MALE.				
Length of right femur	114 mm.
Length of right humerus	85 mm.
NORMAL MALE.				
Length of right femur	96 mm.
Length of right humerus	72 mm.

As a criterion of the relative size of these two animals, taking the pelvis and the skull, the measurements are as follow :—

CASTRATED MALE (PELVIS).				
Extreme distance between iliac crests	56 mm.
Extreme height from the ischial tuberosity to highest part of iliac crest	106 mm.
Antero-posterior diameter of inlet	30 mm.
Transverse diameter of inlet	30 mm.

NORMAL MALE (PELVIS).				
Extreme distance between iliac crests	55 mm.
Extreme height from the ischial tuberosity to highest part of iliac crest	87 mm.
Antero-posterior diameter of inlet	28 mm.
Transverse diameter of inlet	30 mm.

CASTRATED MALE.				
Distance from the anterior margin of the foramen magnum to the premaxillæ behind the posterior, smaller, incisor teeth	80 mm.

NORMAL MALE.				
The same measurement is	79 mm.

Dr. W. O. Merfchejewsky, one of the writers in the appendix¹ to Pelikan's monograph already referred to, shows that the average stature of the Skoptzi exceeds that of the normal male Russian :—

Skoptzi	169 cm.
Normal males	163 cm. 6 mm.
Normal females	153 cm. 7 mm.

The individuals submitted to measurement comprised 22 Skoptzi (aged between 25 and 73), 26 normal males (between 16 and 53),

¹ B. Einfluss der Verschneidung auf die Entwicklung des Männlichen Organismus.

44 females (between 16 and 75); altogether, 92. The difference in stature is due, as the more detailed measurements show, chiefly to an increased length of the tibia (and fibula), the average length of which bone in the Skoptzi is 47 cm. 6 mm., as against 43 cm. 1 mm. in the normal male, and 42 cm. 2 mm. in the normal female. The length of lower limb is strikingly shown in the skeleton of a eunuch from Cairo, presented to the Museum of the Faculty of Medicine, Lyon, and described by Lortet.¹ In a short article in the *Bulletins de la Société d'Anthropologie*, 1901, ii, série 5, a figure of this skeleton is given by Dr. Hikmet and Dr. Regnault. The height of the skeleton is 196 cm.; the humerus is relatively short, the radius and ulna, long; the metacarpals, as well as the phalanges, are elongated; the femur presents scarcely any curvature; the tibia and fibula are of an altogether exaggerated length; the metatarsals and phalanges very long. The elongation affects especially the lower limbs.² Lortet remarks that in the capon it is the legs, and not the wings, which are increased in length; and that the castrated bovine differs from the bull in the elongation of the hind limbs, which straightens up the line of the back, which always drops posteriorly in the uncastrated animal. Godard has also observed that, in the human subject, eunuchs castrated in childhood grow with rapidity at the time of puberty, and mostly acquire a stature of 2 mètres, or even more.

A more striking example of such indirect glandular influence upon the growth of bone than that furnished by the increase in length of the lower limbs after castration, is the well-known one of cretinism, where the deficiency of thyroid secretion is followed by diminished intra-cartilaginous growth of the several bones of the skeleton. And the same result is seen in those cases of cachexia strumipriva, or surgical myxœdema, when total thyroidectomy is carried out in the young subject, should there be no accessory glands in which compensatory overgrowth may take place, as Kocher³ long ago pointed out. Kocher gives a figure of two sisters, of whom the elder had a goitre removed and in whom cachexia strumipriva had developed, the patient being represented as she appeared nine years after the operation.⁴ Although

¹ *Lyon Médicale*, 1896, lxxxi.

² The actual length of the bones of the right limbs in this skeleton are: femur, 53.5 cm.; tibia, 46.3 cm.; humerus, 37.2 cm.; ulna, 32.5 cm.; radius, 30.6 cm.

³ "Ueber Kropfexstirpation und ihre Folgen," *Langenbeck's Archiv für Chirurgie*, 1883, xix.

⁴ Tafel, xii, figs. 15, 16.

the elder, her head reaches only to the shoulders of her younger sister. The significance of this result, however, it must be confessed, is marred by the fact that in the figure of the same sisters, taken together before the date of the operation on the elder, the relative heights are the same.

A question of theoretical interest here arises on which we may briefly digress. Is the striking shortness of the limbs in certain varieties, occurring amongst species of animals in general well proportioned, due to a thyroid deficiency which has become inherited?² The most widely known example of such short-limbedness is the dachshund. Amongst sheep the same thing is illustrated in the ancon short-legged variety, to which Darwin refers.¹ In this case the variety arose *per saltum* as a sport, in a ram lamb. The animal was born in Massachusetts in 1791, and from this one lamb the breed was raised. As these sheep could not leap over fences it was thought that they would prove valuable, but they have been supplanted by Merinos. These sheep were remarkable for transmitting their peculiarity so truly that their original describer, Col. Humphrys,³ never heard of but one—questionable—case of an ancon ram and ewe not producing ancon offspring; when they are crossed with other animals their offspring, with rare exceptions, perfectly resembled either parent, no intermediate forms appearing. Amongst bovines, the example is the Dexter, a variety which appears to be indigenous in the south and south-west of Ireland, and of which a certain number of herds are kept in England.³ The feature of the breed is the shortness of the limbs. The head presents, like that of the dachshund, no cretinoid deviation of form. Now the noteworthy circumstance in connexion with the short-limbed Dexter is the frequency with which it bears cretinous or "bull-dog" calves—a frequency far exceeding anything that obtains in ordinary breeds of oxen. In one herd, for instance, in this country, in the year 1901, out of twenty-one births there were no fewer than seven cretins. This association of an apparently simple short-limbedness in the adults with cretinism in the calves so strongly suggests that the former may be due to a thyroid defect (insufficient to produce the full condition of cretinism, but of a kind capable of producing one of its features) that we were

¹ "Variations of Animals and Plants under Domestication," 1st ed., i, p. 100.

² *Phil. Trans.*, Lond., 1813, p. 88.

³ A full account of these cattle, and of the cretinism so frequently occurring in the calves, has been given by one of us (C. G. Seligmann) in the *Transactions of the Pathological Society*, iv., p. 1.

led to test the effect of prolonged thyroid feeding upon the young dachshund.

As this observation has not hitherto been published we may here record it, although the result was negative. The dachshund, a female, was born on January 1, 1906. It had distemper in April and was convalescent in May. The thyroid administration was commenced on May 10—i.e., when the pup was under five months of age. The batches of tabloids used were freshly prepared (from the dispensary at St. Thomas's Hospital); each contained 3 gr. of dried sheep gland (*Thyroideum siccum*, British Pharmacopœia). For the first week a single tabloid was given each day, during the next week two, then three, and after the fourth week the daily dose was four. No ill effects were at any time observed. The last four tabloids were given on May 12, 1908. The dog was thus kept under thyroid administration, with only an occasional week's intermission, for two years. No elongation of the limbs beyond what is normal to the variety has taken place during the period of growth, and the animal, which is at the present time perfectly well, presents all the marks peculiar to this variety of dog.

We may add to this that the histological details of the ossification at the growing ends of the shafts of the long bones in the dachshund and the cretin are markedly different; indeed, the former present no departure from the normal. Sections were prepared of the lower ends of the radius and ulna and of the upper end of the humerus, of a dachshund pup eleven weeks old, the tissue being removed immediately after death and decalcified in picric acid. The histological picture differs on the two sides of the intermediary zone of cartilage between the shaft and the epiphysis, the latter consisting largely of cancellous bone. On the side towards the shaft the cartilage cells have undergone the typical linear proliferation and form long parallel columns in the long axis of the bone. The calcification of the narrow lines of the matrix between the cell rows, the disappearance of the cells, the irruption of osteoblastic connective tissue from the shaft, and the development of membrane bone upon the calcified lines of the matrix are all in progress exactly as under normal conditions. In this the picture differs widely from that presented by the growing end of the diaphysis in cretinism. Behind the proliferating zone there succeeds a zone of unaltered hyaline cartilage, slightly less in thickness. On the epiphysial aspect of the latter, bone formation is hardly proceeding at all. The cartilage cells for a certain distance are enlarged like those in the proliferating zone on the diaphysial side, and occur in groups of two

and four, but without any trace of a vertical or any other disposition. The method of ossification is much of the same kind, though without active progress. In places, along the line, may be traced the calcification of the matrix between the enlarged cartilage cells, the disappearance of the latter, and the formation of membrane bone upon the remnants of the calcified matrix, which, in place of being disposed in vertical lines, forms a coarse network, incomplete from partial absorption; in other places the irruption of osteoblastic tissue has taken place even less typically, the cartilage being excavated to a slight depth, as it stands, so that groups of cartilage cells remain included in the coarse walls of the primary areolæ, the true bone being laid down on the summits and sides



FIG. 1.

A coronal section of the lower end of the radius of a dachshund puppy, eleven weeks old, which died of distemper. The intra-cartilaginous process of ossification on either side of the intermediary cartilage is normal. That on the diaphysial (upper) side differs from that on the epiphysial in the longitudinal and more active proliferation of the cartilage cells, the growth of the shaft taking place from this aspect. The growth on the epiphysial side is equally normal but less active. In the true cretin the diaphysial process of ossification is as retarded and as scanty as what is shown on the epiphysial side. (Zeiss A; Oc. 4.)

of the upstanding processes of cartilage; and in some spots the layer of membrane bone is continued over the bottom of the recesses themselves. One series of sections was made so as to include the entire thickness of the upper epiphysis of the humerus, nearly the whole of the epiphysis.

consisting of cancellous tissue. In these sections it is apparent that the process of bone production beneath the articular cartilage precisely resembles that in progress on the epiphysial aspect of the intermediary cartilage, and is equally scanty.

It is worth noting that the histological picture presented by this slowly-progressing intra-cartilaginous process of ossification is that seen in cretinism, but in the latter on the *diaphysial* aspect of the intermediary cartilage, as is shown in the sketch made by one of us (S. G. Shattock) illustrating Sir Thomas Barlow's case of foetal cretinism published in the *Transactions of the Pathological Society*, xxxii, plate 33.

There is nothing, then, which makes it a priori unreasonable that the growth of the pelvis accompanying puberty in the human female, and corresponding in time with the maturation of the ovary, may depend upon some function of the latter—not upon the histological maturation and discharge of the ova, but upon the elaboration of an internal secretion of the same class as that which, there is reason to believe, is produced in the case of the male sexual gland, a subject with which we have dealt in the paper already referred to. It is true that in man there is already a differentiation between the male and female pelvis in the foetus, as was first shown by Fehling,¹ and afterwards independently by A. Thomson.² According to Fehling, differences may be detected as early as the third month of foetal life. In the summary of his own paper Thomson writes: "It appears from a consideration of the foregoing facts that during foetal life the essentially sexual characters are as well defined as they are in adult forms, and that any differences that occur during growth between the adult and foetal forms, due, it may be, to the influence of pressure or muscular traction, affect both sexes alike, and that such influences are in no way accountable, as has been maintained, for the characteristic features of the pelvis of the female as contrasted with the male." Nevertheless it remains true that it is not until the advent of puberty that the marked disparity of pelvic growth in the sexes is brought about, and the influence of the ovary may be the effective cause of this, somewhat in the same way that the internal secretion of the testicle brings about the increased growth of hair in the male, not in positions where it is absent in the female, but where it is so little developed as to pass unrecognized.

¹ *Zeitschr. f. Geb. u. Gyn.*, ix and x.

² A. Thomson: "The Sexual Differences of the Foetal Pelvis," *Journ. of Anat.*, 1899, xxxiii, p. 359.

In the human subject observations upon the relationship of pelvic growth at puberty to the maturation of the ovary are wanting, or indecisive, in the case, at least, of Europeans. There is not a single case on record in which (1) both ovaries have been removed in childhood; (2) in which the patient has been traced to womanhood; and (3) in which the pelvis of such a patient has been secured after death and submitted to measurement. All that can be adduced in default of such evidence in the human subject (which alone would be complete) are the occasional observations made in regard to the size of the pelvis during operations carried out in cases of double ovarian disease where the latter, as in bilateral teratomatous cysts, is presumably congenital. Dr. Herbert R. Spencer¹ has recently affirmed that in operating for such a condition in the adult, he has been struck with the contracted dimensions of the pelvis. As these patients were pregnant, however, it is clear the ovaries, one or both, must have been functional. This detracts from the value of the observation, and all that could be concluded at the most would be that the hypoplasia of the pelvis was in some way related to a defective or incomplete development of the ovaries, since it could not have resulted from their full destruction. Dr. Spencer's deduction is based upon the high percentage with which contracted pelvis is associated with ovarian disease (three in fifteen cases).

The Results of Oöphorectomy in the Rabbit.

The first observations we made were carried out upon the Belgian hare. The so-called Belgian hare is a variety of rabbit reared for the market on account of its size. It breeds readily, all through the year; the young mature in about six months. The breed is kept pure in order to maintain its superior size.

In October, 1903, five young Belgian hares were obtained from a dealer in Leadenhall Market; all were supposed to be females. Two were kept as controls.

October 31, 1903: One was spayed. The vagina was exposed by a median abdominal incision, a ligature was placed around it, and it was then cut across above the ligature and the uteri and ovaries removed. The animal was quite well the next day.

November 2: A second animal was operated upon. After abdominal section, it was found to be a male; the testes, long and narrow, lay

¹ Herbert R. Spencer: "Ovarian Tumours complicating Pregnancy, Labour, and the Puerperium," *Surg., Gyn., and Obstet.*, May, 1909. (Read before the American Gynecological Society.)

within the abdomen. A ligature was placed on the spermatic cord above and on the gubernaculum below; the testicle was then excised. The animal was well the next day.

November 9: The uteri and ovaries were removed from a third, exactly as in the case of the first. The animal was well the next day.

The five rabbits, which were kept under healthy conditions and allowed to run about, were killed at the end of July, 1904,—i.e., about nine months afterwards. Of each the skull, the pelvis, and both humeri and femora were prepared by maceration. In each of the two spayed females the ligature was discovered, by dissection, intact on the upper end of the divided vagina. The two intact animals were found on dissection to be females. The measurements taken of the pelvis were as follows: (1) Antero-posterior diameter of the inlet, from the upper margin of the body of the first sacral vertebra to the back of the summit of the symphysis pubis; (2) the transverse diameter of the inlet; (3) the extreme transverse distance between the iliac crests taken from their external borders; (4) the extreme, vertical measurement of the pelvis, taken from the ischial tuberosity to the highest part of the iliac crest. The extreme length of the right femur and of the right humerus was taken in a straight line by means of a sliding centimetric scale, in order to obtain a standard of the size of the different animals.

NORMAL FEMALE.

Inlet of pelvis	Antero-posterior diameter	33 mm.
	Transverse diameter	39 mm.
Distance between iliac crests	58 mm.
Height of pelvis	98 mm.
Length of right femur	105 mm.
Length of right humerus	80 mm.

NORMAL FEMALE.

Inlet of pelvis	Antero-posterior diameter	32 mm.
	Transverse diameter	31 mm.
Distance between iliac crests	54 mm.
Height of pelvis	91 mm.
Length of right femur	97 mm.
Length of right humerus	73 mm.

SPAYED FEMALE.

Inlet of pelvis	Antero-posterior diameter	25 mm.
	Transverse diameter	29 mm.
Distance between iliac crests		58 mm.
Height of pelvis	103 mm.
Length of right femur	108 mm.
Length of right humerus	82 mm.

SPAYED FEMALE.

Inlet of pelvis	Antero-posterior diameter	28 mm.
	Transverse diameter	27 mm.
Distance between iliac crests		50 mm.
Height of pelvis		98 mm.
Length of right femur		103 mm.
Length of right humerus		78 mm.

So far as the females are concerned, these measurements show that in the two intact females both the antero-posterior and the transverse diameters of the pelvic inlet exceed those in the two spayed.

INTACT.

(1)	33 mm. and 33 mm.	...	Length of femur, 105 mm.
(2)	32 mm. and 31 mm.	...	Length of femur, 97 mm.

SPAYED.

(1)	25 mm. and 29 mm.	...	Length of femur, 108 mm.
(2)	28 mm. and 27 mm.	...	Length of femur, 103 mm.

From the length of the femora it will appear that each of the spayed animals was slightly larger than the second one unspayed; and one of the spayed was larger than the larger of the two intact.

The Results of Oöphorectomy in the Pig.

The next animal we selected was the pig. In the Natural History Collection at South Kensington there are the skeletons of two examples of *Potamochoerus*, the pelves of which we have measured, with the following results:—

1364 B: Male, fully grown. Symphysis pubis synostosed; no epiphysal lines on the long bones. Pelvis: Maximum transverse diameter of inlet, 80.5 mm.; antero-posterior diameter, taken from the summit of the symphysis to the sacral promontory, 103 mm. Femur: Length taken by means of callipers from the summit of the head to the lowest point of the inferior articular extremity, 206.5 mm; this bone was measured as an index to the size of the animal.

1363 B: Female, fully grown. Symphysis pubis synostosed; no epiphysal lines on the long bones. Pelvis: Maximum transverse diameter of inlet, 80.5 mm.; antero-posterior diameter, taken from the summit of the symphysis to the sacral promontory, 104.5 mm. Femur: Length, taken as in the preceding specimen, 182.5 mm.

The length of the femur in the female of these two specimens is 24 mm. less than in the male—i.e., the animal is somewhat smaller—nevertheless, the diameters of its pelvis are equal to those of the larger male.

These measurements were sufficiently promising, as indicative of a sexual differentiation in the pelvis, to lead us to take the common domesticated pig for studying the results of oöphorectomy.

In October, 1903, three piglets were selected from the same litter: one female was kept intact, one was spayed, one male was castrated. Nothing untoward occurred after the operations, which were done by an expert "cutter," the whole of the uterus, with the ovaries, being removed through a small abdominal incision, and the testicles by an incision through each half of the scrotum. The animals were killed, when full grown, November 2, 1904, December 6, 1904, January 20, 1905, and of each, the pelvis and right femur were prepared by maceration. The measurements in the case of the two sows are as follow:—

SPAYED SOW.

Antero-posterior diameter of inlet of pelvis	93 mm.
Transverse diameter of pelvic inlet	65 mm.
Extreme distance between the outer borders of iliac crests	166 mm.
Height of pelvis from ischial tuberosity to the summit of the iliac crest	236 mm.
Length of right femur from summit of the great trochanter to lowest point of condyles	206 mm.

UNSPAYED SOW.

Antero-posterior diameter of inlet of pelvis	91 mm.
Transverse diameter of pelvic inlet	64 mm.
Extreme distance between the outer borders of iliac crests	163 mm.
Height of pelvis from ischial tuberosity to the summit of the iliac crest	231 mm.
Length of right femur from summit of the great trochanter to lowest point of condyles	194 mm.

It will appear that all the measurements of the pelvis of the spayed slightly exceed those of the unspayed animal. The differences in diameters of the inlet of the two pelvises do not amount to more than an excess of 2 mm. on the antero-posterior and of 1 mm. on the transverse. The femur of the spayed is 12 mm. longer than that of the unspayed, showing that the animal was the larger. This reduces the very small differences in the pelvic diameters to *nil*. In the castrated boar the antero-posterior diameter of the pelvic inlet is 85 mm.; the transverse is 65 mm.; the extreme length of the right femur is 202 mm. As one

effect of castration is to increase the length of the long bones, very little can be deduced from the above measurements, as the standard of size is thus vitiated. The transverse diameter of the pelvic inlet is equal to that of either of the sows; the antero-posterior is less.

The differences brought out in the pig being, like those obtained from the rabbit, so small as to be indecisive, it became imperative to make use of an animal of much larger dimensions, and one in which there is normally a pronounced difference between the pelves of the two sexes. It is probably true generally that in animals (such as the rabbit and pig) which bring forth a large number at a birth there is but little pelvic differentiation, whereas in those which bear but one or two young the size of the foetus would, as compared with the parent, be relatively larger; and this would entail an increased capacity of the true pelvis on the part of the female.

The Results of Oöphorectomy in the Ox.

These considerations led us to test the question upon the bovine. In the British Museum (Natural History) there are the skeletons of a full-grown male and female European bison and those of a fully-grown Chillingham bull and Chillingham cow. The following measurements, which we were enabled to make, bring out a difference in the size of the pelvis in the two sexes of these cattle:—

EUROPEAN BISON (*Bos bonassus*).

Male (fully grown).

Pelvis	Transverse diameter of inlet	163 mm.
	Antero-posterior diameter in inlet	224 mm.
Femur (length taken by means of callipers)	435 mm.

Female (fully grown).

Pelvis	Transverse diameter of inlet	171 mm.
	Antero-posterior diameter of inlet	247 mm.
Femur (length taken by means of callipers)	396 mm.

CHILLINGHAM OXEN (*Bos taurus*).

Male (fully grown).

Pelvis	Transverse diameter of inlet	146 mm.
	Antero-posterior diameter of inlet	204 mm.
Femur (length taken with callipers)	351 mm.

Female (fully grown).

Pelvis	Transverse diameter of inlet	163 mm.
	Antero-posterior diameter of inlet	219 mm.
Femur (length taken with callipers)	330 mm.

116 Shattock & Seligmann: *Oöphorectomy and the Pelvis*

In these four skeletons it will be found from the measurements given that in each of the females the diameters of the pelvis exceed those of the males, although the length of the femur is less:—

		Male (bison)		Female	
Pelvis	Transverse diameter ... °	...	163 mm.	...	171 mm.
	Antero-posterior diameter	...	224 mm.	...	247 mm.
Femur (length)	435 mm.	...	396 mm.
		Male (Chillingham)		Female	
Pelvis	Transverse diameter	146 mm.	...	163 mm.
	Antero-posterior	...	204 mm.	...	219 mm.
Femur (length)	351 mm.	...	330 mm.

On April 27, 1906, a calf was spayed (from the flank) by Mr. F. L. Gooch, F.R.C.V.S., of Stamford, Lincolnshire. The animal was all red in colour, of the Shorthorn breed, and was exactly 7 weeks old when the operation was carried out. Recovery was uninterrupted, the external wound healing by first intention. The parts removed comprise the ovaries, together with the Fallopian tubes, and a portion of each uterine cornu 5 cm. in length; where cut across the cornu has a diameter of 0.6 cm. Each ovary is 2 cm. in the longer diameter; in the periphery of the divided surface there are a series of follicles (the largest 2 mm. in diameter), the presence of which gives a moruloid character to the free, uncut exterior.

The animal was shortly afterwards transferred to the Elstree farm of the Lister Institute, where it was kept until August 4, 1908, when it was killed, being at that date 2 years and 5 months old, and in perfect general condition. The teats were diminutive; they measured in length not more than 2 cm., and were correspondingly reduced in diameter. Each of the mammary glands was represented by a huge mass of fat. The pelvis was taken out entire, together with the several bones of the right hind limb; and the skull, with its short, slightly curved horns, was likewise kept. Before being killed the height of the animal was taken with a vertical measuring rod and sliding cross-piece furnished with a spirit-level. The height from the withers (the highest point between the shoulders) to the ground was a fraction over 12 hands (48½ in.). Measured with a tape from the withers over the trunk and down the fore-limb, the distance was 50 in. From the withers to the root of the tail the distance, with the tape, was 51 in. The distance, with the tape, between the centres of the rounded eminences formed by the crests of the ilia was 17 in. The maceration of the pelvis was first conducted so as to leave the bones connected by their ligaments; the pelvis was allowed

to dry, and was measured. The maceration was afterwards carried to completion and the bones disarticulated.

MEASUREMENTS OF THE PELVIS OF THE SPAYED ANIMAL.

Antero-posterior diameter of the inlet	19.8 cm.
Transverse diameter of the inlet	14.7 cm.
Extreme distance between the iliac crests, taken from the outer borders	41.3 cm.
Extreme distance between the ischial tuberosities, measured from the inner side	14.3 cm.

The bones of the right hind-limb, after maceration, placed in natural apposition and in the extended position, gave a total length of 46 in. (116.8 cm.). In November, 1908, Mr. Gooch was able to supply us with the corresponding parts of a fully-grown, intact cow, 6 years old, of the same breed—viz., Shorthorn, red. The animal was suffering from double pneumonia and pleurisy. The measurements, taken a day or so before it was killed, were: Height, just over 50 in.; length, from withers to root of tail, 50 in.; width between hips, 18 in.; the animal was just 1 in. higher over the hips than over the withers. After maceration, the pelvis of the unspayed animal gave the following measurements:—

Antero-posterior diameter of the inlet	24.0 cm.
Transverse diameter of the inlet	21.5 cm.
Extreme distance between the iliac crests, taken from the outer borders	56.0 cm.
Extreme distance between the ischial tuberosities, measured from the inner side	20.1 cm.

The bones of the right hind-limb after maceration, placed in natural apposition and in the extended position, gave a total length of $49\frac{5}{8}$ in. (126.4 cm.). There is thus a difference in the length of the right hind-limbs, that of the spayed animal being $3\frac{5}{8}$ in. (9.6 cm.) shorter than that of the unspayed. When the diameters of the pelvic inlet are worked out in proportion to the length of the limb the results are as follow:—

Length of limb of unspayed : Antero-posterior diameter of inlet of unspayed ::
Length of limb of spayed : Antero-posterior diameter of inlet of spayed

$$126.4 : 24 :: 116.8 : x$$

The proportionate antero-posterior diameter in the spayed animal would be 22.2. As a matter of fact, this diameter of the pelvis in the spayed animal is only 19.8 cm.

Length of limb of unspayed : Transverse diameter of inlet of unspayed ::
Length of limb of spayed : Transverse diameter of inlet of spayed

$$126.4 : 21.5 :: 116.8 : x$$

The proportionate transverse diameter in the spayed animal would be 19.8 cm. As a matter of fact, this diameter of the pelvis in the spayed animal is only 14.7 cm. There is a pronounced difference in the distance between the ischial tuberosities as measured from the inner side. Taking, again, the proportion between the limbs as a standard, the distance between the tuberosities in the spayed would be 18.5 cm., whereas it is really only 14.3 cm. And, lastly, there is a pronounced difference in the width between the iliac crests. Taking the proportion between the limbs as a standard, the distance between the crests in the spayed animal would be 51.7 cm., whilst it really is only 41.3 cm. The proportion between the antero-posterior and transverse diameters of the inlet, moreover, differs. In both, the former exceeds the latter (contrary to the case in man), but the excess in the unspayed is less than in the spayed—i.e., the transverse diameter is relatively greater than the antero-posterior.

	SPAYED.				
Antero-posterior diameter of inlet	19.8 cm.
Transverse diameter of inlet	14.7 cm.
	UNSPAYED.				
Antero-posterior diameter of inlet	24.0 cm.
Transverse diameter of inlet	21.5 cm.

If the proportions obtaining in the spayed are calculated out for the unspayed, the antero-posterior diameter of the unspayed—20 cm.—would be associated with a transverse diameter of only 17.8 cm., as against the actual 21.5 cm. This observation upon the bovine demonstrates, therefore, that the growth of the pelvis is reduced as a result of double oöphorectomy when carried out at an early age.

Amongst the Skoptzi sect, already referred to, the mutilations carried out upon the female are limited to the external genitals, the mammæ and nipples; removal of the ovaries is not practised. In India it is stated that ablation of the ovaries in the young is a time-honoured custom, and that in the adults the growth of pubic hair is wanting, the mammæ undeveloped, the vaginal orifice practically obliterated, and the pubic arch narrow. This statement appears in Gould and Pyle's "*Curiosities of Medicine*." The authority given for it is Knott (*Medical Press and Circular*, London, 1860, ii, p. 33). A search in the College Library has failed to discover Knott's article in the place cited or elsewhere.¹

¹ Dr. Norman Chevers, in his well-known "*Manual of Medical Jurisprudence for India*" (3rd ed.), gives no information relative to this subject.

The effect produced by the function of the ovary upon the growth of the accessory organs and the pelvis may be ascribed, *mutatis mutandis*, as in the male, to the formation of an *internal* secretion by the sexual gland and its distribution through the body, as distinguished from the *external*—viz., the discharge of the ova. Whether the interstitial cells play a part in conjunction with ovulation is a question parallel to that presented in the case of the male. As we have said¹ in connexion with the testicle, as concerned in the production of secondary sexual characters: "The function of spermatogenesis, although not itself the whole or sufficient cause, may be the initial factor of a dual or even a more complex process. It is quite within the bounds of possibility that certain of the epithelial cells within the tubuli may produce a pro-secretin such as is produced within the intestinal epithelium; that the chemical changes accompanying spermatogenesis in other of the cells of the tubule may lead to the conversion of this pro-secretin into a secretin, much as the acid chyme does in the case of the pro-secretin present in the intestinal cells; and that the secretion so formed may, without being shed into the lumen of the tubule, be transferred to the lymph spaces, and thus eventually reach the general circulation and incite those metabolic changes in distant parts of the body which disclose themselves as secondary sexual characters." "In regard to the interstitial cells of the stroma, they have characters so unmistakably glandular that some secreting function, probably a sexual one, must be assigned to them, and they may, of course, take a part in the elaboration of such a secretion as that suggested." And so in the case of the female. Ovulation *per se* may not suffice to bring about the result; nor is it easy to see how the mere discharge of ova alone could produce such widespread effects. Yet there are the epithelial cells lining the follicle and reflected over the ovum itself, and beyond this the interstitial cells in the fibrous wall of the follicle and in the general stroma. And such an internal secretion, it is conceivable, may be evolved by the combined action of two or more sets of these cells. The undergrowth or hypoplasia of the accessory organs, after castration or oöphorectomy, is possibly the result, not of the absence of a stimulating substance acting directly upon the various tissues of these organs, but rather of a diminished or unaugmented blood supply to the several structures concerned, brought about through the intervention of the nervous system. We are led back, indeed, to a modified hypothesis of trophic centres

¹ *Trans. Path. Soc.*, lvi, p. 78.

the action of which is excited or withheld by the selective affinity of a circulating substance, and which determine hyperplasia or hypoplasia by inducing, or failing to induce, an increased supply of blood. The co-ordination of the growth of the several parts is fundamentally chemical, though executed through the intervention of the nervous system.

The Pelvis in Eunuchs.

This subject, from its connexion with the foregoing, demands consideration, for there are current statements, and even mensurations, which may be taken to imply that the pelvis of the eunuch comes to resemble that of the female. Gilbert White,¹ in his thirty-second letter, has it: "Eunuchs have smooth, unmuscular arms, thighs, legs, and broad hips, and beardless chins and squeaking voices." That externally there is a suggested increase in the dimensions of the pelvis is indisputable; the important question is whether this is apparent or real. Seeing that the removal of the ovaries prevents the female pelvis from assuming its proper dimensions, it would be an extremely paradoxical result if the loss of the testicles was followed by the very result brought about in the female only by the function of the ovaries. Such a result, did it obtain, would give support to the view, adopted by some, that each individual is essentially bisexual; and that on the loss of the male gland, e.g., the female characters become spontaneously evolved, and vice versa. That the testicle is responsible for the development of the positive masculine characters is clear, from the sequences of castration. It would be necessary, however, to assume that the testicle was responsible, in addition, for the latency or occultism of those feminine characters which are of a positive as distinguished from a negative kind, the latter being simply persistencies of such as are puerile,—the presence of the testicle would, in some way, inhibit the growth of the breasts and of the pelvis, whilst at the same time it induced the masculine growth of the hair on the face, the increase in the size of the larynx, and the growth of the prostate and the other accessory organs. So that on the removal of the testicles not only would the positive male characters fail to appear, or, if present, would retrogress, but such female features as are positive in kind would become substituted for them. This would imply, so it might be imagined, the production by the testicle of specific "restraining

¹ "Natural History of Selborne," edited by Bowdler Sharpe, 1900.

substances" antagonistic to the growth of the structures referred to. The other hypothesis, and the one to which we ourselves have hitherto inclined, is that which regards the positive male, or female, characters as dependent upon the function of the sexual gland; and to view cases of sexual transformation as indicative of the retrogression, say, of ovarian tissue, and the activation of testicular tissue hitherto dormant.¹

Before the latter theory can be dismissed, it would be necessary to prove the entire absence of a second sexual tissue in the individual in whom a sexual transformation, complete or partial, has taken place. So far as the pelvis is concerned, it has yet to be determined to what extent the feminine appearance ensuing after early castration may not be due to the local obesity which results. In an appendix to Pelikan's work on the Russian Skoptzi (*loc. cit.*), Dr. W. O. Merfchejewsky furnishes four figures bearing upon this question. In fig. 2 is shown an adult from whom the genitalia had been removed at the age of 6 years; in fig. 3 an adult deprived of his genitalia at the age of 13 years. In these there is an obvious increase in the breadth of the hips, and the shoulders appear relatively narrow; the general configuration recalling that of the opposite sex. Both these individuals, however, exhibit a marked local obesity of the thighs and hips, and this obesity may conceivably account for the whole of the apparent increase of the pelvis. The series of measurements given by the author named are, it must not be forgotten, all of them taken from the living. And, indeed, they prove too much; for they not only show that the "pelvis" of the castrated is larger than that of the normal male, but larger than that of the normal female in the particular directions in which the normal female exceeds the male. Thus the circumference is 2 cm. in excess of that of the normal (Russian) female; the bitrochanteric distance is 2 cm. in excess; the external conjugate diameter of the pelvis, taken from the spinous process of the fifth lumbar vertebra to the symphysis, is 1 cm. beyond that of the female; the distance between the anterior superior spinous processes of the ilia exceeds that in the female by 1 cm. Normally the bi-iliac diameter (from the most prominent point of the outer border of one iliac crest to the other) is greater in the male than in the female: in the Skoptzi this distance measures 1 cm. over the normal Russian female, and offers, therefore, no anomaly.

¹ S. G. Shattock and C. G. Seligmann: "Hermaphroditism in the Fowl," *Trans. Path. Soc.*, lvii, p. 69.

The local obesity of eunuchs is one of their well-known features. Dr. J. McClean, of the British Hospital, Constantinople, informs us that, in the case of the eunuchs attending in the harems, the scrotum, with the testicles and the penis, are cut away, the mutilation being carried out at the age of from 10 years to 16 years, and that the individuals put on fat over the buttocks and thighs so as to resemble the female; the obesity increases with age. Sir Harry Johnston states that in Negro eunuchs from the Sudan he has noticed a tendency towards a "lateral steatopygy," or a coxal lipomatosis, as it might be more accurately designated.¹

It becomes of paramount importance, therefore, to deal directly with the skeleton, and this is precisely where the evidence is meagre. The eunuch skeleton from Cairo described by Lortet (*loc. cit.*) has a height of 196 cm.; in life the individual would have been above 199 cm. The increased stature is due chiefly to the exaggerated length of the tibiae. Now, of this skeleton it is stated that the pelvis was very small, almost atrophic; the foramina ovalia were very large, but left between them a very narrow pubic symphysis.² The photograph of this skeleton shows, indeed, nothing suggestive of the female type in the pelvis.

In the next place we may describe the pelvis of the eunuch whose pelvic viscera have been referred to in the earlier part of this communication, and which is now in the Museum of the Royal College of Surgeons, to which it was presented by Professor A. F. Dixon, of Dublin. The height of the individual himself (whose mother was Chinese and father British) was 5 ft. 5½ in. The measurements of the pelvis, of which the bones retain all their natural ligamentous connexions, are as follow:—

Between the most prominent points of the external borders of the crests of the ilia	29.5 cm.
Between the outer borders of the anterior-superior spinous processes of the ilia	25.5 cm.
Between the last lumbar spine and the front of the symphysis pubis	17.9 cm.
Antero-posterior diameter of inlet	9.2 cm.
Transverse diameter of inlet	12.9 cm.
Transverse diameter of outlet	8.7 cm.
Sub-pubic angle	60°

¹ S. G. Shattock: "Steatopygy," *Proc. Roy. Soc. Med.*, 1909, ii (Path. Sect.), p. 207 *et seq.*

² This inaccurate expression means, presumably, that the body of each os pubis was narrow—i.e., of the male character.

For purposes of comparison we may take the measurements of the normal European pelvis as given in the third and last edition of Professor Cunningham's "Anatomy":—

FALSE PELVIS.					Male	Female
Bi-iliac	28.2 cm.	27.3 cm.
Interspinous	24.0 cm.	25.0 cm.
Conjugate diameter	17.6 cm.	18.0 cm.

TRUE PELVIS.					Male	Female
Antero-posterior diameter of inlet	10.1 cm.	11.0 cm.
Transverse diameter of inlet	12.7 cm.	13.5 cm.
Transverse diameter of outlet	8.8 cm.	11.0 cm.

An analysis will show that in the false pelvis the bi-iliac distance, which is normally greater in the male than in the female, is 29.5 cm., against the average 28.2 cm. of the male. The interspinous is 25.2 cm., against the female 25 cm.¹ and the male 24 cm. In both of these dimensions the eunuch pelvis exceeds that of the average male. The conjugate diameter is 17.9 cm. against the female 18 cm. In the true pelvis the antero-posterior diameter of the inlet is male, being 9.2 cm., against 10.1 cm. male and 11 cm. female. The transverse diameter of the outlet is male, being 8.7 cm., against 8.8 cm. male and 11 cm. female. The transverse diameter of the inlet exceeds that of the average male, being 12.9 cm., against 12.7 cm. male and 13.5 cm. female. This is the only measurement in the eunuch's pelvis which really approaches the female. In the actual specimen it is 13.2 cm., but as the skeleton is a "natural" one, with the interpubic disk and the sacro-iliac synchondroses intact, though dried, it is not too much to subtract 3 mm. from the actual transverse diameter of the inlet to bring the measurement in line with pelvis artificially articulated; it presents, moreover, an anomalous want of symmetry, the pelvic cavity being more extensive on the left side than on the right. When we study the characters of this pelvis in detail, it is unmistakably of the male type: The pubic arch is narrow, with an angle of 60°; the descending rami are thick and everted; the body of the os pubis wants the squareness of this part in the female—i.e., it is much narrower from side to side than in the horizontal direction; the transverse diameter of the first sacral centrum is 5.2 cm., and the breadth

¹ This measurement is stated in Quain's "Anatomy," 10th ed., to be normally greater in the male than in the female.

of each lateral mass is 3·2 cm., which measurements are, in the female, quite or nearly equal to one another—i.e., the cross-diameter of the centrum equals the breadth of the two lateral masses combined. Again, notwithstanding the fact that the bi-iliac diameter is above the usual male average, the masculine divergence or “splay” of the false pelvis is fully pronounced, and is altogether unfeminine; the vertical buttress on the outer aspect of the ilium, passing from the crest to the acetabulum, has the highly pronounced character it has in the male. The horizontal diameter of the right, undiseased acetabulum is 5 cm., and the vertical 5·1 cm. Now, the diameter of the head of the femur in the female never exceeds 4·5 cm., its size in the female being not relatively, but absolutely, less than in the male. And finally, the sacro-sciatic notch is typically masculine, for in the female the upper, horizontal limb is equal to, or exceeds, the vertical, whereas in the eunuch the upper of its limbs is 3·2 cm. and the lower 5·3 cm.

As a third observation upon the pelvis of the eunuch: In the preparation of the pelvic organs of the Skoptzi in the Museum of the Royal College of Surgeons, already referred to, the anterior portion of the pelvis has, fortunately, been preserved. It shows the same narrow pubic arch as in the case of the Chinese eunuch, the highest part of it being fully occupied with the corpus spongiosum and crura of the corpora cavernosa; the body of the os pubis shows, moreover, the same want of the feminine squareness.

These three observations made upon the pelvic skeleton, then, furnish no evidence to show that the pelvis of the male, after castration carried out presumably before puberty, acquires the female characters.

We may append, in conclusion, two views of the pelvis of the Chinese eunuch, which will graphically illustrate the correctness of this provisional deduction (figs. 2 and 3).

FIG. 2.



A front view of the pelvis of the Chinese eunuch described. It has been photographed in the horizontal position to show the narrowness of the pubic arch and the want of feminine squareness in the body of the os pubis. There is a considerable osteo-arthritic lip to the left acetabulum, and, on the right side, to the upper and lower margins of the fifth lumbar centrum and the first piece of the sacrum. Although the distance between the iliac crests is above the average, the masculine divergence of the ilia or "splay" of the false pelvis is fully pronounced and altogether unfeminine.

(Museum of the Royal College of Surgeons.)

FIG. 3.



A lateral view of the same pelvis, photographed in the natural oblique position. It shows the masculine character of the sacro-sciatic notch, the upper, horizontal limb of which is 2 cm. shorter than the lower: in the female the limbs are of equal length, or the horizontal exceeds the vertical. The vertical buttress passing from the crest of the ilium to the acetabulum, also, has the pronounced development it presents in the male.

(Museum of the Royal College of Surgeons.)

Pathological Section.

February 15, 1910.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

Attempts to produce Chondromatous or Osteomatous Growths by the Grafting of Fœtal Bones.¹

(Interim Report.)

By S. G. SHATTOCK, C. G. SELIGMANN, and L. S. DUDGEON.

OF all the tissues of the body that may be selected for the purpose of experimentally producing a tumour, cartilage offers perhaps the greatest advantages and the best prospect of ultimate success. Not only is the cartilage of foetal or of growing bone readily obtainable, but it is a particularly hardy and resistant tissue, which thrives upon a very scanty blood-supply, or, indeed, without any immediate vascularity whatever. Its capacity for growth, again, as witnessed at the epiphysial ends of the long bones, is extensive, even under natural conditions. And the recognized origin of chondromata and osteomata in connexion with foetal or growing remnants of precursory cartilage makes it probable that such tumours might be more easily produced by experiment than those of a higher histological structure. The grafting or transplantation of foetal cartilage with this object has been carried out by not a few since Cohnheim advanced his suggestive theory of the origin of new growths. The results, nevertheless, have been so uniformly negative that it may be taken as definitely proved that the mere transference of foetal cartilage into the body of a second animal of the same, and *a fortiori* of another, species is fruitless.

¹ The expenses connected with this work were defrayed by a grant from the Imperial Cancer Research, which we here very gratefully acknowledge.

At the present time no one has succeeded in experimentally producing either a malignant or a benign tumour. When the abnormal growth has arisen "spontaneously" or "naturally," as is said for want of better knowledge, it may be transferred by grafting, but it will be obvious that this is relatively a small achievement compared with what the experimental origination of either an innocent or a malignant tumour itself would be. In the endeavour to raise a benign tumour, then, some new departure is necessary: something, it is clear, is wanting in the simple methods already tried.

In November, 1907, we ourselves had formulated the following hypothesis as a basis of the present work, which we not long afterwards commenced. The question resolves itself, so it seemed to us, into a determination of what factors normally restrain or limit the growth of any single one of the many tissues of the body, and adapt it to the needs of the rest. How is the growth of the various structures co-ordinated? The fundamental element in the co-ordination is most probably a chemical one. The theory we arrived at was that each of the tissues furnishes a "restraining body," the whole of which "bodies" in conjunction inhibit the unlimited growth of any one tissue in particular. For example, the cartilage of the body (like each of the other tissues) tends, we may assume, *per se* to grow indefinitely, and without limitation. But against this inherent endeavour, each of the tissues other than cartilage furnishes a restraining substance, the sum of which substances limits the production of this particular tissue, and co-ordinates its growth with that of the rest; and the same would be true, *mutatis mutandis*, of any individual histological structure: the growth of any one in particular is restrained by the combined products of all the rest. The hypothetical substances we may name "restraining bodies," "corpora cohibentia."

They are not lysins; for the unlimited growth of any given tissue is not checked by the lytic destruction of an actual surplus formation of it; but they are bodies of a distinct order, the existence of which can be deduced by the biological phenomena actually witnessed in the body. Two theoretical methods of overcoming or neutralizing the action of the "restraining bodies" offer themselves. Selecting cartilage, which is the tissue made use of in our present experiments:—

(1) The resistance might be broken down or exhausted by the frequently repeated and voluminous introduction of the foetal or growing cartilage. Just as the natural immunity to a bacterial infection may not withstand the introduction of a bacterium in very large amounts (i.e., the

introduction of quantities that would never obtain under natural circumstances), so it might be in the case of a grafted tissue. The fowl is naturally immune, in this sense, to tetanus; yet the disease can be induced by the insertion of excessive doses of the bacillus. White mice, again, are naturally immune to glanders. But as one of us¹ showed, the lesions of the disease may be produced by the subcutaneous injection of a sufficient quantity of a recently raised potato-culture.

(2) The restraining or anti-bodies might be neutralized by means of anti-restraining or anti-antibodies.

The memorable result obtained by Bashford,² which shows that an extract of mouse-skin will inhibit the growth of a subsequent graft of mouse-epithelioma, is not so much at variance with the hypothesis propounded as it might at first appear. For it may be supposed that the restraining bodies to the unlimited growth of skin, furnished by all the other tissues in conjunction, are selected by, and loosely fixed in, the skin itself; in which they are present, therefore, in greater amount than elsewhere.

In these experiments made by the use of skin, we must presumably understand epidermis, since in the graft it is the epithelium which is the essential element transferred; the stroma of the new growth is furnished by the recipient. A similar prophylaxis, as Bashford has shown,³ may be brought about by the subcutaneous injection of mouse-blood, but in a less notable degree. The probable explanation here, as Bashford holds, is that the blood contains the same substance as does the skin, but in less amount. In harmony with the working hypothesis already advanced, the restraining body to the unlimited growth of skin furnished by the several tissues other than skin, is in sufficient amount in the circulating blood to produce a like inhibitory result, but, being less in amount than that accumulated in the skin from the blood unceasingly circulating through it, the result is less pronounced.

The second method of attempting to raise a simple or malignant tumour we do not take up here, as our investigation in this direction is not yet sufficiently advanced.

In the work we now put on record, we have adopted the new departure of grafting foetal bones, not on a single occasion only (which experience has proved fruitless as a means of inducing the growth of a chondroma), but of grafting such bones in regular succession, and for

¹ S. G. Shattock, *Trans. Path. Soc. Lond.*, 1898, xlix, p. 335.

² Bashford, Murray and Haaland, *Berl. klin. Woch.*, 1907, xlv, pp. 1194, 1238.

³ Bashford and Murray, *Brit. Med. Journ.*, 1906, ii, p. 209.

an indefinite period, with the object of breaking down the action of the restraining bodies which theoretically keep the growth of the general cartilage of the body within limits, and adapt it to the requirements of the organism as a whole. Furthermore, in order to aid the desired result, we have grafted the foetal bones (1) into young—i.e., growing—rabbits, (2) into pregnant does. For as in the first the bones of the skeleton are themselves growing, we were interested to discover how the body would behave in restraining the growth of grafted bones—bones, that is to say, which might be regarded as additamentary to those of the general skeleton. And in the second, as the cartilage and bones of the foetuses *in utero* are growing without hindrance on the part of the mother, foetal bones grafted during pregnancy into another part of the body might perchance themselves grow without restraint, at least for the same period as that of the gestation. Of the second experiment we made as we proceeded, the important modification, moreover, of grafting the bones of the foetuses of the doe herself into the subcutaneous tissue, for reasons which will be presently given.

It will be obvious (to judge from analogy) that the repeated introduction of growing cartilage beneath the skin of a second animal, in place of exhausting the resistance to its growth, might strengthen it; the production of the specific restraining body might be increased. In order to test this possibility, we propose to use the blood periodically of one of these animals as an intraperitoneal injection to another rabbit, with a view of producing an anti-restraining body in the latter, the presence of which might conceivably allow of foetal cartilage grafted into the second animal to grow.

TECHNIQUE.

The foetal bones were obtained by arranging for a periodic supply of pregnant does. These were killed by a blow on the back of the neck. The uteri were immediately exposed, removed from the body, slit up, and the foetuses withdrawn, one by one, and kept in the incubator at 37° C.¹ The necessary number of limbs were cut away and placed in a capsule of warm salt solution, kept in a water bath at 37° C. The whole of the procedures were carried out with strict aseptic precautions, all the instruments and apparatus used being boiled, as well as the salt solution. Under salt solution at 37° C., the skin was stripped from the

¹ In some cases the foetuses were of about three weeks; in most they were near full term, or about four weeks, the normal period of gestation in the rabbit being about thirty-one days.

limbs with two pairs of forceps, and the muscles torn away, the bones being then disjoined and carefully picked clean, so that no soft parts were left in connexion with them other than the periosteum and shreds of the articular capsules, which were too firmly attached to be pulled entirely away. The salt solution was from time to time changed, and the bones, in this way, washed free of surface blood. They were inserted into the subcutaneous tissue by means of a cannula specially devised for the purpose. The bones having been inserted lengthwise, one or two at a time, into the end of the cannula, were then pushed through it by means of a solid metal plunger. The site of puncture was finally sealed with cotton wool and collodion. In no case did anything untoward, either immediately or remotely, follow the procedure.

THE GRAFTING OF FŒTAL BONES INTO YOUNG RABBITS.

RABBIT I.

July 8, 1908.—A young rabbit received subcutaneously about a dozen foetal bones from a pregnant doe just killed.

July 23.—It received six foetal bones, including two scapulae.

The animal died on August 24 with coccidiosis. The grafted bones were removed and are preserved in the Museum of the Royal College of Surgeons. They are shown in the accompanying photograph (fig. 1).

RABBIT II.

June 24, 1908.—A young rabbit received subcutaneously two foetal femora and two foetal humeri.

July 1.—About a dozen foetal bones were inserted beneath the skin.

July 23.—Six foetal bones were inserted.

September 16.—Eight foetal bones (humeri and femora) inserted.

October 14.—Four foetal bones inserted.

October 28.—Four foetal bones inserted.

November 30.—Three foetal bones inserted.

January 8, 1909.—Four humeri inserted.

January 29.—Four humeri and two scapulae inserted.

Summary of Rabbit II.—This animal received subcutaneously, between June 24, 1908, and January 29, 1909, inclusive, nine insertions of foetal bones, many of them whilst it was still growing. It was killed in the autumn of 1909. The several groups of bones were found loosely encapsulated beneath the skin. Their epiphysial ends had undergone an obvious enlargement.

RABBIT III.

September 16, 1908.—A young rabbit received four foetal bones beneath the skin.

October 14.—Received four foetal bones.

October 28.—Received four foetal bones.

November 30.—Received four foetal bones.

December 11.—Received four foetal bones.

January 8, 1909.—Received four foetal humeri.

January 29.—Received four foetal humeri and scapulae.

Summary of Rabbit III.—This animal received subcutaneously, between September 15, 1908, and January 29, 1909, inclusive, seven insertions of foetal bones, many of them whilst it was still growing. It was killed in the autumn of 1909. The several groups of bones were found loosely encapsulated beneath the skin. Their epiphysal ends were enlarged.

THE GRAFTING OF FETAL BONES INTO PREGNANT DOES.

Of the two animals used in these experiments, one received on the earlier occasions a certain number of the bones from foetuses obtained by killing other pregnant does, but since then she has been receiving only the bones of her own foetuses. The other doe has received throughout only the bones of her own progeny. The foetuses under these circumstances have never been used except they were found alive: they were killed by a blow on the back of the head, *seriatim*, as required. The remaining foetuses of the litter were destroyed. In these cases the additional precaution was adopted of sterilizing the *exterior* of the limbs after their removal by immersing them for a short while in warm carbolic-acid solution (1 : 20); they were then washed in warm salt solution and transferred to a capsule of the same (kept at 37° C.), in which the skin was stripped off; and thence to a second capsule of warm salt solution, in which the muscles were torn away with two pairs of forceps, as already detailed. Sometimes the foetuses were obtained within a few minutes of birth, at others after the lapse of a few hours.

Our preference for the bones of the foetuses born of the doe herself to those of the foetuses of another rabbit, arose from the fact that in the course of certain grafting experiments carried out upon birds¹ we were impressed by the ease with which detached fragments of testicle would grow within the abdomen of cockerels from which the testicles

¹ S. G. Shattock and C. G. Seligmann, *Proc. Roy. Soc.*, and *Trans. Path. Soc. Lond.*, 1905, lvi, p. 57.



FIG. 1.

The foetal bones which were grafted, at two sittings, into the subcutaneous tissue of a young rabbit (I). One group lies on a portion of the thoracic wall of the host, two of the ribs of which will be recognized, the bones having been implanted, in part, into the muscular substance. The cartilaginous epiphyses of the several bones have grown since their insertion, in some cases so as to form bulbous swellings at the ends of the shafts. The enlarging ends have, as yet, undergone no calcification. (Natural size.)



FIG. 2.

One of the bones isolated by dissection from a group inserted beneath the skin of one of the two preceding rabbits. The several bones were firmly united into a compact mass by new-formed connective tissue. In that isolated, the enlargement of the articular ends is more pronounced than was observed in the case of any of the other animals as yet killed. Each of the ends is densely calcified. The shaft has undergone no increase in circumference or in length. (Natural size.)

(having been unintentionally ruptured during removal) had been incompletely taken away; and by the negative result attending all attempts to graft the testicle from one bird into another of the same variety, whether capon or hen. Allusion was made (*loc. cit.*) to the difficulty of completely removing the testicles in cockerels, and to the fact that a considerable number of birds from which apparently all testicular tissue had been removed by castration, subsequently developed peritoneal grafts which became spermatogenic, and caused the bird to assume the full male characters. So readily did this process of grafting from the glands of the birds themselves take place on to the peritoneum, that the intra-abdominal insertion of testicular tissue was practised upon other birds, mainly with the object of seeing whether their sexual characters could be by this means experimentally restored or altered. The grafts were made into capons, into young and old hens, and into hens from which the oviduct had been removed. The last-mentioned experiments were carried out since it is a current belief that in young birds in which the oviduct has been cut across or removed, the development of the ovary is prevented and the bird rendered sexless.¹

In no case did the grafted fragments of testicle grow, or produce any change in the external sexual characters of the bird into which they were introduced. This failure to obtain a positive result can hardly be explained by lack of vitality in the grafted tissue, or by any inflammatory reaction in the peritoneum of the bird receiving it. The technique was devised, at least, with the object of eliminating such contingencies. The bird to receive the graft was given an anæsthetic, and the feathers removed from the front and sides of the abdomen, and lower portion of the thorax. The skin having been cleansed, an incision was made to one side of the middle line. Meanwhile the neck of the bird which was to provide the graft was wrung, and the skin rapidly stripped from the body; the abdomen was widely opened and the testicles removed with sterile instruments. In a few of the experiments they were placed for a short space in salt solution, which had been boiled and cooled to 37° C., in a small beaker standing in a basin of warm water in which a thermometer stood at from 37° C. to 40° C.; but in most cases the testicles were used immediately they were removed. Whichever course was followed, the glands were held over the opening in the abdomen of the anæsthetized fowl, and small pieces, cut off with scissors, were allowed to drop directly into the peritoneal cavity within which they were distributed with the

¹ W. B. Tegetmeier, "Poultry," 3rd ed., p. 131. This belief we (S. G. Shattock and C. G. Seligmann) have proved by experiment to be fallacious.

finger. The intestines were easily held aside, and at no time were they extruded; the wound was finally sutured and sealed with collodion. In some instances the grafted tissue was sutured to the parietal peritoneum.

In every case the birds made a rapid and uneventful recovery. In a few the insertion was repeated, but invariably with the same result. In no instance did the grafted tissue grow or live in its new environment; and on killing the birds at varying times after the operation, either no trace of it was found or it was represented by one or more encapsulated necrotic masses.

These results demonstrate how different is the history of detached fragments of the male sexual gland in birds, when they are implanted into the abdominal cavity of the individual in which the gland has developed, and when the tissue is implanted into a second bird, although one of the same variety. The experiment of grafting the cock's testicle into the abdominal cavity of the hen was originally made by Hunter (whose preparations are in the College of Surgeons), and with a like negative result. A similar want of success has attended the transference of thymus tissue from one animal to another of the same species.¹ The animals used were kittens, rabbits and puppies, those furnishing the graft being of the same litter as those receiving it. The insertion was made into the peritoneal cavity, but in every case the tissue failed to live.

The following are the details of the experimental history of the two pregnant does, beneath the skin of which a regular succession of foetal bones are being grafted :—

WHITE DOE I.

March 22, 1909.—Had young; the six humeri of three of the foetuses were inserted beneath the skin of the mother.

April 29.—Had young in the night; foetuses found dead and not used.

June 11.—Had young; eight humeri inserted subcutaneously.

July 21.—Had young; eight humeri and six scapulae, detached from the former, inserted beneath the mother's skin.

October 13.—Had young; seven humeri were inserted beneath the skin.

November 20.—Had young; as they were found dead, no use was made of them.

January 26, 1910.—Had young; eight humeri inserted beneath the skin. In order to facilitate the access of nutritive fluid to the proliferating cartilage, the broad lower end of each bone was split vertically with scissors.

March 4.—Had young; eight humeri inserted beneath the skin. The end of each was split.

¹ Experiments on the grafting of the thymus gland in animals. L. S. Dudgeon and A. E. Russell, *Trans. Path. Soc. Lond.*, 1905, lvi, p. 238.

Summary.—Up to the present time this doe has been pregnant on eight occasions, and has received as grafts bones of her own fœtuses on six occasions. The grafted bones are all loosely encapsulated beneath the skin in groups as they were inserted, but no tumour has, so far, grown in connexion with any of them.



FIG. 3.

A group of fetal bones which were grafted beneath the skin of the back of a young sandy doe on May 14, 1909. The animal had young for the first and only time in August, 1909, and was killed November 8, 1909. The bones comprise six humeri of the fœtuses of a pregnant doe, which was killed near the full term for the purpose of supplying them. The epiphysial ends, especially the upper, of the several bones are notably enlarged. The shafts have not increased either in circumference or in length. The ends themselves are still cartilaginous, as told by piercing them with a fine needle. This experiment is not further referred to in the text. (Natural size.)

BLACK DOE II.

October 14, 1908.—Received four bones of the fœtuses of another rabbit which was killed for the purpose of obtaining the fœtuses before delivery. She herself had young on October 25.

October 28.—Received four bones from the fœtuses of another rabbit killed for the purpose.

November 30.—Received four bones from the fœtuses of another rabbit killed for the purpose.

December 11.—Received four bones of the fœtuses of another rabbit killed for the purpose. On January 18 she herself had young. On January 27 the animal was skiagraphed.

January 29, 1909.—Received four humeri, with the scapulae, of the fœtuses of another rabbit killed for the purpose.

Up to this time the doe had been twice pregnant, and had received as grafts the bones of the fœtuses of other rabbits on five occasions. From this date onwards the grafted bones were obtained solely from her own progeny.

March 29.—Had young; received six humeri subcutaneously from her own foetuses.

May 6.—Had young; received six humeri from her own foetuses.

June 17.—Had young; received eight humeri from her own foetuses.

July 30.—Had young; received six humeri from her own foetuses. The upper ends of the radius and ulna were left in connexion with some of the humeri.

October 6.—Had young, which, when seen, were dead.

November 18.—Had young; received humeri from her own foetuses.

January 1, 1910.—Had young; received eight humeri from her own foetuses.

In this case the flat lower end of each bone was split longitudinally with scissors in order to allow direct access of the nutritive fluid to the proliferating cartilage. On January 17 the doe was skiagraphed.

February 1.—Had young, which, when seen, were dead.

March 10.—Had young; received eight humeri from her own foetuses. The lower end of each bone was split.

Summary.—Since March 29, 1909, inclusive, this animal has been nine times pregnant, and has received subcutaneously, as grafts, bones of her own foetuses on seven occasions. Previously to this she had received bones from the foetuses of other rabbits (killed for the purpose) on five occasions; and had been herself pregnant twice, but without her own foetuses having been made use of. The grafted bones are all loosely encapsulated beneath the skin, in groups as they were inserted, but no tumour has so far grown in connexion with them, neither have they grown continuously in their new situation as they would have done in the rabbits from which they were obtained, and this notwithstanding the occurrence of repeated pregnancies on the part of their mother.

In the case of both these does, within a few days of the grafting the buck has been put with the doe; copulation has almost directly taken place, and pregnancy has almost regularly ensued from this date. The young have been born on or about the thirty-first day; bones of a certain number of the foetuses have been isolated, and inserted subcutaneously in the manner already detailed; pregnancy has been again induced, and after the birth of the resulting foetuses, the same procedures have been carried out. This regular repetition has only been broken in cases when the young have been born at an unseasonable time, and found dead. This, however, has happened but few times in the case of the black doe. The various groups of transplanted bones become loosely encapsulated in the subcutaneous tissue, and can be readily slipped about beneath the skin, or raised with it.

We may here introduce two skiagrams which were kindly taken for us by Dr. A. H. Greg in the X-ray Department of St. Thomas's Hospital. They show the same group of bones in the subcutaneous tissue between

the shoulders of the black doe. One skiagram was taken in January, 1909; the other in January, 1910. It will be seen that no change has taken place in the interval in the size of the bones, which were in this particular instance obtained from foetuses not the offspring of the rabbit herself.

At the present time these two does are being kept regularly pregnant, and are receiving as grafts the humeri of their own foetuses, six or eight at a sitting. The experiment we propose to prolong indefinitely, and not to kill the animals even after the grafting should be discontinued,



FIG. 4.

A skiagram taken January, 1909, showing a group of foetal bones grafted beneath the skin of the black doe (II), the bones in question being those of foetuses not her own. (Natural size.)

but to keep them alive in order to see whether at some future period growth will take place from the dormant cartilage.

In connexion with the grafting of osteogenic cartilage, or rather cartilage which is precursory to and determines the development of bone, the influence of the thyroid gland must not be lost sight of. That the internal secretion of the thyroid in some manner aids in the normal growth of the bones is obvious from the fact that the intracartila-

ginous growth of bone is markedly reduced as a result of thyroid deficiency. The action of the secretion upon the intracartilaginous growth of the bone may be viewed as due to a stimulation; or (to interpret it according to the restraining hypothesis already advanced) the thyroid secretion may act by neutralizing the bodies which normally restrain the growth of bone, so that in its absence the restraint is unchecked, and the growth of the bones correspondingly curtailed. The same influence would appear, also, to hold after the completion of growth.



FIG. 5.

A skiagram taken a year later than the preceding, and showing the same group of grafted bones. They have undergone no change in size. The deep shadow given by the epiphysal ends is probably due to their calcification, a result observed in the case of the grafted bones examined in certain of the other cases. (Natural size.) (The skiagram is taken from the other side, the bones being consequently reversed.)

In the rabbit, after experimental fracture of the metatarsal bones and ribs, the growth of callus is reduced if the animals have been previously subjected to thyroidectomy.¹ Led by such considerations, we have now commenced the administration of thyroid to the white

¹ "Ueber den Einfluss des Schilddrüsen-Verlustes auf die Heilung von Knochenbrüchen." Inaugural dissertation, Maurice Steinlin (University of Lausanne, 1899).

doe (I), which has received only the bones of her own fœtuses. The gland is being given in the ordinary form of tabloids made with dried sheep thyroid. We are not using rabbit thyroid, as it is clear from what is known of the action of sheep thyroid in cases of human myxœdema and cretinism that the source of the secretion is immaterial.

We have, in conclusion, to acknowledge the assistance kindly given to us by Mr. H. A. F. Wilson in connexion with certain parts of this work.

Examination of the Brain of a Man who Lived Seven Hours after Receiving a Shock of 20,000 volts.¹

By F. W. MOTT and EDGAR SCHUSTER.

THE material was sent to the Pathological Laboratory, Claybury, for examination by Sir Thomas Oliver on January 12, 1909. It consisted of a small piece of brain showing a small hæmorrhage, the medulla oblongata, and portions of the spinal cord. The following notes accompanied the material, which was preserved in alcohol:—

The notes which we have been able to obtain are extremely scanty. The patient was admitted to the Newcastle Infirmary, and the doctor who sent the case reported that the man had received a shock of 20,000 volts; he lived seven hours after the injury. There were several burns, one large one over the occipital region laying bare the bone of the skull. The post-mortem examination was made thirty-nine hours after death. At the autopsy a hæmorrhage was found in the occipital lobe. In addition to the cerebral hæmorrhage there were petechial hæmorrhages in the visceral pericardium, and the urine in the bladder was deeply blood-stained.

The fluid in which the material came was deeply stained with blood. The portions of tissue were placed in alcohol, and subsequently in xylol, then embedded in paraffin, and sections cut upon the rocker microtome $5\ \mu$ in thickness. These were stained by the Nissl method and with polychrome and eosin.

Microscopic examination of the sections showed the following histological changes which are represented in the accompanying drawings made by one of us (Edgar Schuster).

¹ Read at the laboratory meeting of the Section at the National Hospital, February 1.

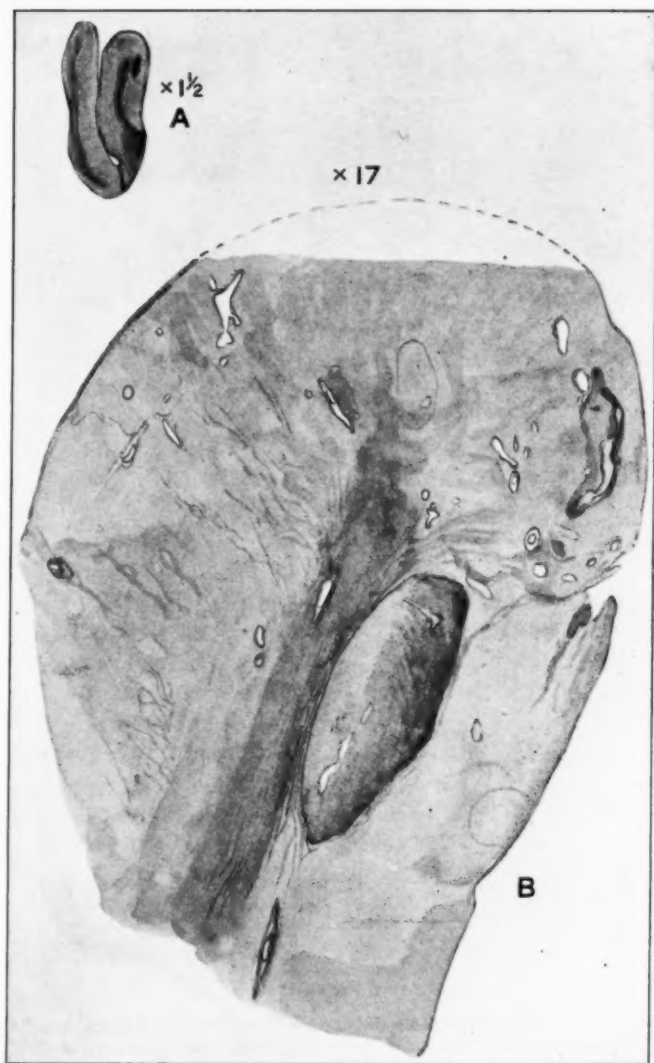


FIG. 1.

A, Small section of occipital cortex, showing a hemorrhage about 2 mm. long. Magnification $1\frac{1}{2}$. B, The same magnified 17 times.

The hæmorrhage is in the cortex, and is typical of many other hæmorrhages in the cerebral cortex; it is probable that these small vascular hæmorrhages were similar to the petechial hæmorrhages in the pericardium; they were very probably due to the effect of the electric

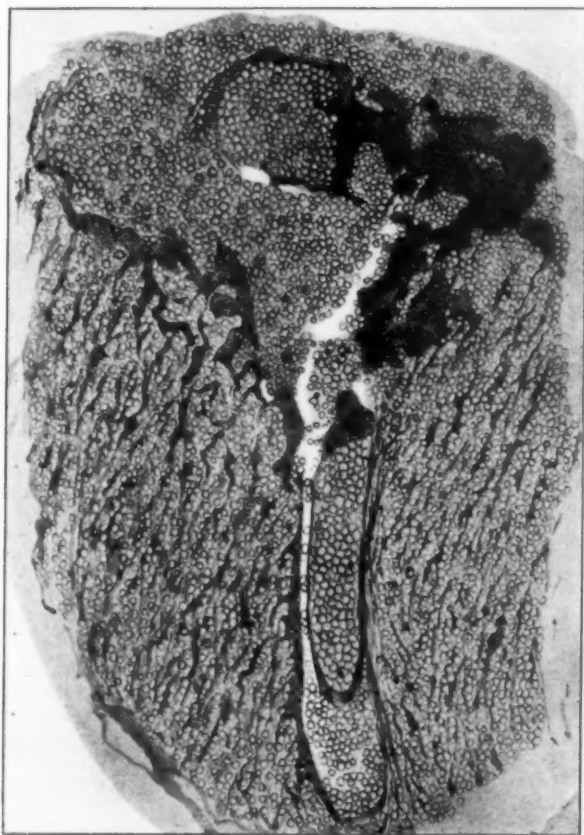


FIG. 2.

Small hæmorrhage into the cortical substance; masses of coloured amorphous substance are seen between the corpuscles, indicating hæmolysis. Magnification 310.

shock on the blood, for we observed in places not only extravasation of corpuscles as seen in fig. 2, but sometimes, instead of corpuscles, irregular masses of amorphous-coloured matter, as if the corpuscles had

undergone hæmolysis. Such a condition would account for the blood-stained urine. In fig. 3 there are seen masses of coloured amorphous substance between the corpuscles. The ganglion cells are uniformly stained a diffuse dull purple, as if they had undergone a change of the

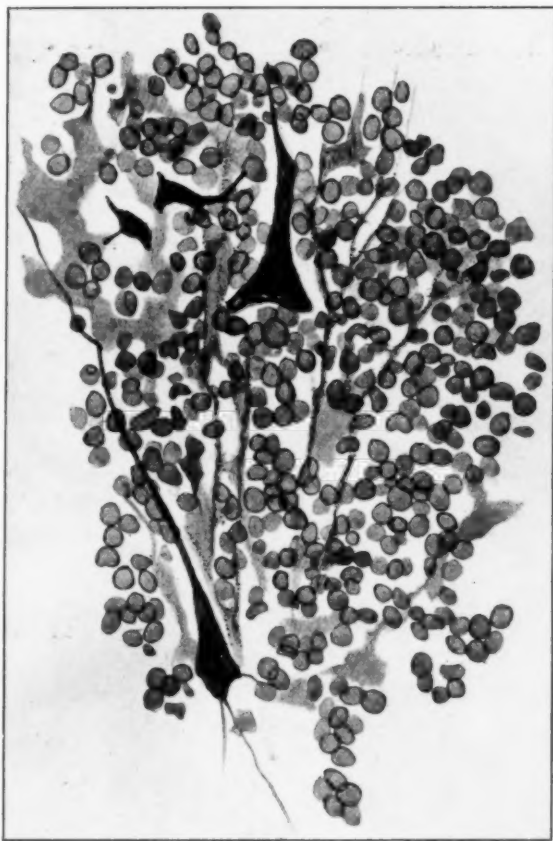


FIG. 3.

Small hæmorrhage of cortex showing several ganglion cells exhibiting a diffuse staining reaction. Between the corpuscles are coloured amorphous masses. Magnification 640.

nature of early coagulation necrosis. The pyramidal cells (fig. 5) elsewhere in the cortex than in the actual hæmorrhages exhibit a change which was found also in the cells of the medulla oblongata (fig. 4)

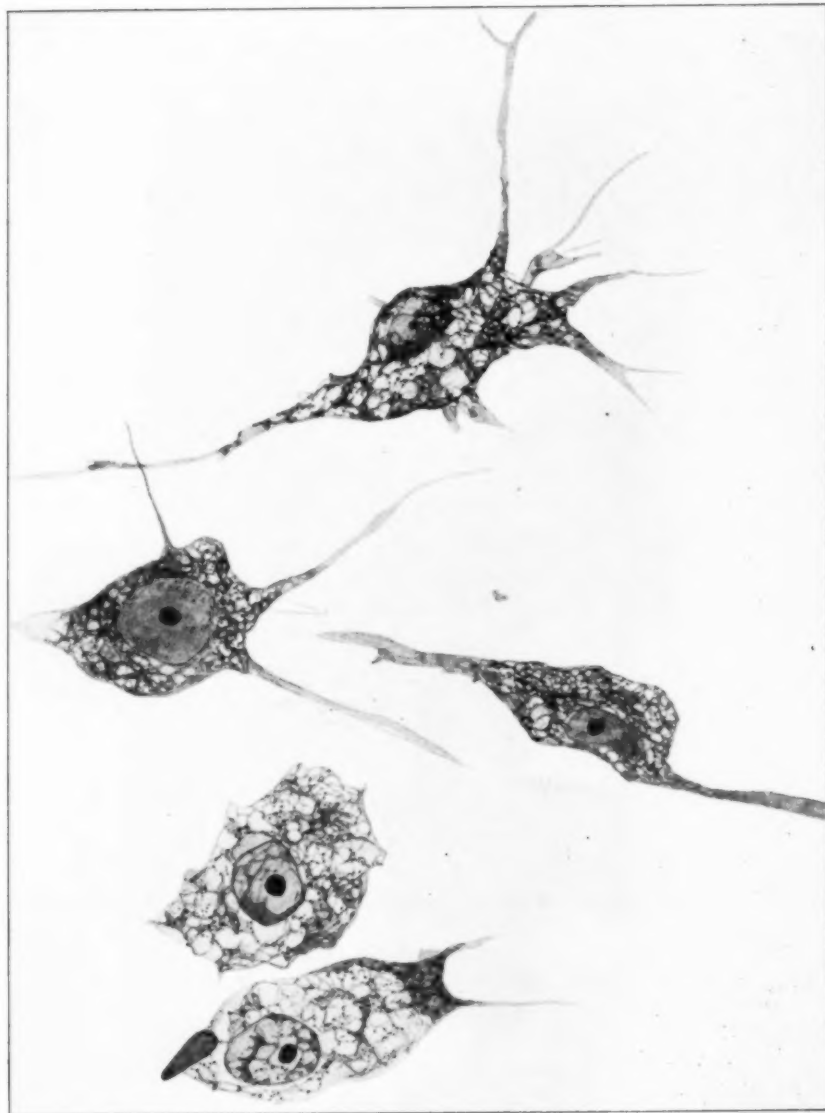


FIG. 4.—Four cells from the medulla oblongata, showing a very diffuse chromatolysis and thereby revealing the intracellular and intranuclear networks. Polychrome staining. Magnification 810.

and the spinal cord. It is one of marked diffuse chromatolysis whereby an intracellular and intranuclear network is disclosed, owing to hardly any of the basophile chromophilous substance being left. That which remains is more or less encrusted on the trabeculae of the network. I have never seen so profound and universal a chromatolysis, and we

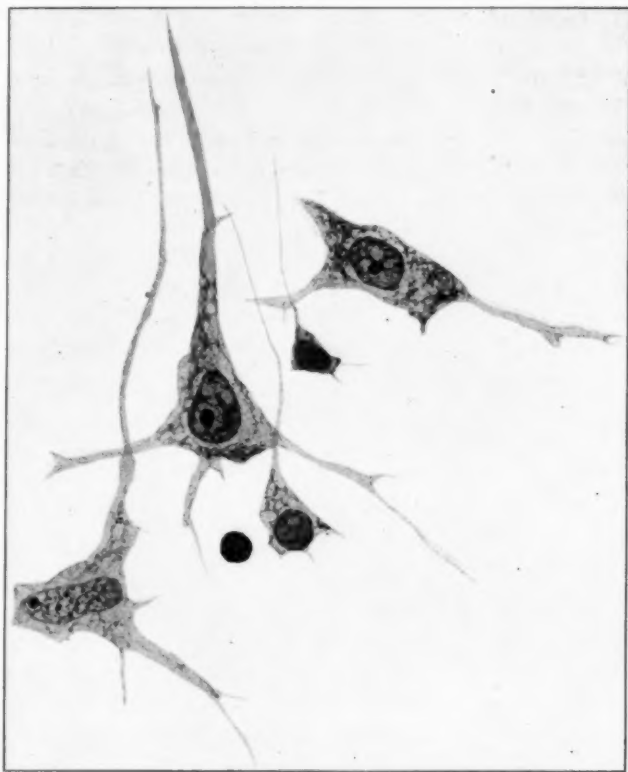


FIG. 5.

Cells of cerebral cortex, showing the same diffuse chromatolysis as the cells of the medulla oblongata. They likewise reveal, in the same manner, the intranuclear and intracellular networks. Magnification 810.

would suggest that it was either directly or indirectly the result of the electric shock. It might be said that the changes may be post-mortem effects, but we have never seen changes like this occur in the ~~x~~medulla

oblongata from post-mortem effects, even though the examination was not made till three days after death; moreover, the accident occurred in the middle of winter. If, then, we are permitted to exclude the changes due to post-mortem decomposition, how can we explain these chromolytic changes? The question may be asked: Did the electric shock directly affect the nervous tissue in such a way that the cells could no longer assimilate material during the seven hours the man lived, and consequently the neurones used up the basophile substance without any subsequent reparation? Or was this change due partly or wholly to the effect on the blood and the circulation? It may be suggested that the electric current not only destroyed the osmotic membranes of a large number of the red corpuscles, thus permitting the hæmoglobin to escape, but at the same time it also destroyed the osmotic membranes of the nerve-cells.

Pathological Section.

March 15, 1910.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

A Case of Bile-producing Primary Malignant Tumour of the Liver ("Malignant Adenoma"): with Remarks on the Cells of Malignant Tumours retaining some of the Metabolic or other Functional Characteristics of the Cell-type from which they are derived.

By F. PARKES WEBER.

THE patient, F. R., a man aged 69, was admitted into the German Hospital on January 27, 1910. He said that he had enjoyed good health, and gave no history of previous illnesses of importance. But since the summer of 1909 he had suffered from increasing debility, with loss of flesh. He did not, however, consult a doctor until six weeks before admission, when he complained of pain and swelling in the abdomen. In the hospital the patient was found to have ascites; the liver could be felt enlarged and hard, its surface somewhat uneven, and its lower edge reaching to the umbilical level. The spleen could not be distinctly palpated, but the splenic dullness was enlarged. The patient's temperature was slightly above the normal, but never as high as 100° F. The pulse was about 80 to 90 per minute. Although there was no obvious jaundice of the skin or conjunctivæ, a trace of bilirubin was detected in the urine, which likewise contained a little albumin. The patient died on February 3, 1910.

At the post-mortem examination the peritoneal cavity was found to contain much ascitic fluid mixed with blood, intraperitoneal hæmorrhage having evidently taken place from a vein passing over a large mass of new growth in the right lobe of the liver. The *liver* (weight, 89 oz.) was much enlarged, and had an uneven surface owing to the bulging caused by the presence of multiple tumours. There was no perihepatitis. The gall-bladder and extra-hepatic bile-ducts were free. The extra-hepatic blood-vessels were not thrombosed. On section the organ was seen to be moderately cirrhotic, but to consist chiefly of rounded nodules, or masses of new growth, varying from the size of a pea to double the size of a medium apple. The largest mass, in the right lobe, might possibly have been the primary growth. The tumours were almost entirely necrotic, having been transformed into a pulpy substance ("purée cancéreuse"), coloured greenish or yellow by the bile. In fact, nearly the whole of the liver substance was replaced by these pultaceous masses, somewhat of the consistence of mashed potato. It is quite possible that some of the pultaceous masses included necrotic liver tissue as well as necrotic new growth. The original cirrhotic liver substance was still fairly well preserved at the margin of the left lobe. In addition to the appearances already described there were a few hæmorrhages. The spleen (weight, 15 oz.) was enlarged and enclosed in a greatly thickened capsule, evidently the result of old perisplenitis; in fact, the spleen might be termed a "Zuckergussmilz," just as the German term "Zuckergussleber" has been applied to livers whose capsules are greatly thickened from old perihepatitis. After a careful examination no evidence of new growth could be found elsewhere than in the liver, with the exception of a single cherry-sized tumour-nodule (evidently metastatic) in the left lung. Macroscopic and microscopic examination of the rectum showed the presence of hæmorrhoids, but not of any new growth. The prostate gland was likewise examined macroscopically and microscopically for evidence of new growth, but with negative result. The heart (weight, 8½ oz.) was hypoplastic, and the pericardial surfaces were adherent from old pericarditis. There were pleuritic adhesions on both sides of the chest. The kidneys (weight of both together, 9 oz.) showed a little old interstitial fibrosis and a condition of cloudy swelling. Nothing of special interest was discovered in the pancreas, adrenals, stomach, or rest of the alimentary canal. The lymphatic glands (microscopic examination) were apparently not infiltrated with new growth. I have to thank my house physician, Dr. G. Dorner, for his great help in the examination of the case.

Microscopic Examination of the Liver.—The minute non-necrosed nodules of new growth (fig. 1) consist of cells more or less resembling those of the hepatic parenchyma, and showing a marked "trabecular"



FIG. 1.

Photomicrograph ($\times 97$) of the border of one of the tumour-nodules to show the "trabecular" arrangement (in tubular columns) of the cells. The new growth occupies the upper part of the field, the liver tissue the lower part.



FIG. 2.

Drawing of tubular columns of tumour-cells enclosing inspissated bile-pigment. From the same section as fig. 1, but under higher magnification.

arrangement (the "*épithéliome trabéculaire*" of Hanot and Gilbert¹); that is to say, they form tubular columns of cells more or less imitating the columnar arrangement of the liver cells in the normal hepatic acini. The columns in the new growth are, however, much thicker than the normal columns of liver cells, and are more irregular in their arrangement, and the tumour cells stain slightly more deeply (hæmatoxylin and eosin) than the normal liver cells. Some of the tubules contain inspissated bile (fig. 2), which is coloured dark green in the sections stained by hæmatoxylin. In some parts the columns of cells in the new growth are separated from each other by considerable spaces containing connective-tissue shreds. These "intertubular" spaces were doubtless originally much smaller, and were occupied by imperfectly formed blood-vessels, but have become enlarged during the process of preparation of the sections. Some portions of the new growth (probably on the point of becoming necrotic) show hæmorrhagic and small-cell infiltration. The tumour-capsules, if any can be said to exist, are formed by the compressed surrounding tissue. The liver substance shows small cell infiltration, chiefly of interacinous distribution, and a moderate degree of cirrhosis; there are likewise scattered hæmorrhagic areas.

REMARKS.

The case is doubtless one of a form of primary carcinoma of the liver in which the tumour cells are derived from the glandular bile-secreting cells of the hepatic parenchyma. After a careful search (partly microscopical) no primary growth could be discovered in the stomach, rectum, intestines, prostate, &c.² The primary hepatic nature of the growth is, moreover, abundantly confirmed by the appearance of the tumour cells and by their ("trabecular") arrangement in tubules, some of which enclose inspissated bile. The malignant nature of the tumours is proved not only by their great development in the liver, and by the necrotic changes they have undergone there—doubtless due to involvement of the hepatic blood-vessels—but also by the presence of a

¹ V. Hanot and A. Gilbert: "*Études sur les Maladies du Foie*," Par., 1888, p. 41.

² In all apparently primary cancers of the liver the importance of careful search for a possible primary growth elsewhere is well known. In regard to examination of the rectum it is also a recognized fact that small tumours of the rectum may give rise to large secondary growths in the liver. Thus, in 1904, a man (aged 50) under my care died from secondary carcinoma of the liver. The organ in question was of immense size and weighed 12½ kg., that is to say, over one-fifth of the total body weight (60½ kg.), whereas the primary growth in the rectum formed a projecting lump of the size only of a large cherry.

secondary nodule in the lung, which, however, I regret, was unfortunately not examined microscopically.

The disease in question has been variously termed "primary carcinoma with cirrhosis" (Hanot and Gilbert), "cirrhosis maligna," or, as H. D. Rolleston (on the analogy of carcinoma of the stomach supervening on chronic gastric ulcer, and primary carcinoma of the breast supervening on chronic interstitial mastitis) prefers to call it, "cirrhosis carcinomatosa." The tumour has also been spoken of as "hepato-adenoma" (Lancereaux), "adeno-carcinoma of the liver," and "malignant adenoma of the liver" (H. Ribbert). By analogy with terms which have been suggested or employed to designate primary tumours of other organs (e.g. "hypernephroma," and possibly also "myeloma" and "splenoma") the term "hepatoma"¹ or "malignant hepatoma" might also be adopted for these primary carcinomata of the liver. If, however, this principle of nomenclature were to be carried further, the text-books would soon be containing descriptions of "mastoma," "nephroma," "pancreatoma," and possibly also of various forms of "gastroma" and "enteroma."

Do the cells of malignant tumours retain some of the metabolic or other functional characteristics of the type of cell from which they are derived?

In the present case the tumour cells were certainly secreting bile (or at all events bile-pigment) in some parts, as shown by the inspissated secretion enclosed within many of the tubular columns of the growth. It is quite possible that a stage of simple adenoma or "nodular hypertrophy" of liver cells precedes the development of the malignant adenoma (primary carcinoma) of the liver cells, and it might perhaps be objected that bile is only secreted by the cells during the "nodular hypertrophy stage," but that when the stage of malignancy is reached, the tumour cells are no longer bile-producing. It is quite likely, indeed, that the power of secreting bile gradually diminishes when the tumour becomes malignant, and in connexion with this question I am exceedingly sorry that the metastatic tumour in the lung of the present case was lost. However, H. D. Rolleston² figures a section from a case of hepatic "carcinoma with cirrhosis" in which the tubules of the growth contain still more inspissated bile than in

¹ Vide L. Renon, E. Géraudel and R. Monnier-Vinard, "L'hépatome—tumeur primitive du foie," *Presse médicale*, Par., 1910, xviii, p. 29.

² Rolleston, "Diseases of the Liver," 1905, p. 477; see also F. J. Wheeler, *Guy's Hosp. Rep.*, 1909, lxiii, pp. 225-241.

the present case; and H. Ribbert,¹ in a recent article on "malignant adenoma" of the liver, refers to bile-production by the tumour cells as a pathological distinction of great importance for the recognition of this class of new growths. In this connexion one may call to mind that some of the primary tumours of the bone-marrow termed "multiple myeloma" (whose malignancy is evidenced by the bone-erosion which they cause, and occasionally by involvement of lymphatic glands) have been found to consist of cells whose cytoplasm contains granules more or less resembling those in normal myelocytes.² From the same point of view it is likewise interesting that certain tumours of the hypernephroma type in children have apparently given rise to a precocious plethoric type of obesity (or to excessive growth of the skeletal muscles) and premature sexual development.³ I need scarcely refer to the well-known (though rare) metastatic tumours (in bones, &c.) of typical thyroid structure.

One may furthermore instance the well-known production of "cell-nests" ("epithelial pearls") in some of the metastases from squamous-celled carcinomata of the skin and tongue as well as in the primary growths. Perhaps improved methods of chemical and physiological histology will ultimately show that the cells of certain primary malignant tumours of the breast, pancreas, and other organs do retain some of the functional characteristics of the cells of the organ from which they are derived.

¹ Ribbert, *Deutsch. med. Wochenschr.*, 1909, xxxv., p. 1607.

² Cases of F. P. Weber (1903), Sternberg (1903), Charles and Sanguinetti (1907), A. Herz (1908), and Ueber (1908). The references to the literature on these cases are given by Weber and Ledingham, *Proc. Roy. Soc. Med. (Path. Sect.)*, 1909, ii, p. 206.

³ See Bulloch and Sequeira, *Trans. Path. Soc. Lond.*, 1905, lvi, p. 189; and Guthrie and Emery, *Trans. Clin. Soc. Lond.*, 1907, xl, p. 175.

Amyloid Degeneration in an Adenoma of the Liver.

By S. G. SHATTOCK.

De degeneratione amyloide in hepatis adenomate.

SUMMARIUM.

Tumor ipse magnitudine insignis, in hepatis substantiâ, circumscriptus, continetur.

Observatae sunt in neoplasmatibus degenerationis species diversae—adiposa, mucinosa, colloides, hyalina, calcarea—hoc, autem est, quoad scio, primum tumoris exemplum cum degeneratione amyloide affecti.

E lobulis constat illis vix dissimilibus hepatis ipsius, de cellulis magnis, cum oleo saepe distentis, ac sine stromate, constructis, et in vasorum cancellis capillarum dispositis.

Arteriolae venulaeque in telae connexivae septis sustentae, tumidae sunt et vitreae; atque in sectionibus microscopicis idonee tinctis (methyl violet; iodine) colorem degenerationis amyloidis proprium monstrant. Similiter affectum est rete capillare inter tumoris cellulas vagatum, in gradibus autem diversis in diversis lobulis.

In hepate ipso extra tumorem, degenerationis notae amyloidis obviae sunt. E malariâ, degenerationis forsitan causâ, aegrotaverat homo.

THE tumour itself is remarkable in the first place for its size, if the mere volume of a growth can be said to possess an interest; for it is the largest of its kind on record, with the exception of one which was removed during life from a patient who was admitted later into St. Thomas's Hospital, and to which I may refer at the same time. The matter of real interest in the tumour, however, is the amyloid degeneration with which it is affected. Seeing that every other form of degeneration is to be met with in new growths—fatty, mucinoid, colloid, hyaline, calcareous—the occurrence of amyloid has ever presented itself to me as a possibility which observation was sure, in the course of time, to demonstrate; and, in the tumour under consideration, this expectation has at length been realized. This is, I believe, the first instance in

which amyloid degeneration has been discovered in a new growth. The macroscopic preparations (one of which is in the museum of St. Thomas's Hospital and the other in that of the Royal College of Surgeons) show each, a slice of an oval tumour $6\frac{3}{4}$ in. (17 cm.) in its longer diameter, with the portion of the liver in which it has grown. Over the greater part of the tumour the hepatic substance is so thinned as to be unrecognizable, but at one pole it forms a concavo-convex cap measuring, where thickest, 1 in. (2.5 cm.). The growth itself is sharply circumscribed and thinly encapsulated. In addition to a coarser, incomplete subdivision into lobes, the divided surface presents a more minute lobulation, corresponding in places not unclosely with that of the liver itself. The colour of the tumour, in preparations made after the Kaiserling method and preserved in 50-per-cent. glycerine, is uniformly pale, but in a few places it is blotched with dark brown or black.

The specimen was obtained from a man, aged 27, who was admitted into St. Thomas's Hospital under the care of Dr. Sharkey, October 24, 1904. He was discharged shortly afterwards, and subsequently died in the Lambeth Infirmary. The patient could not recall having had any illness as a child. There was no history of syphilis, but a fairly pronounced one of alcoholism. He went to India in 1895. In 1900 he suffered from "dysentery"; he passed blood in his stools and was in hospital six weeks. In 1901 he had ague. In 1902 a diagnosis of dilatation of the stomach was made, and he was invalided home and admitted into Netley Hospital. The diagnosis then made was disease of the liver; this organ was enlarged and painless, the enlargement being palpable chiefly in the epigastrium and left hypochondrium. As the possibility of hydatid suggested itself, an exploration was carried out on December 20, 1903. The liver was found enlarged, smooth, and elastic; it was aspirated without result. The day following the patient "coughed up 6 oz. of tuberculous-looking pus," but there was no alteration in the symptoms. He left Netley Hospital in March, 1904; since then he had felt very weak, but beyond this there was no other complaint. When admitted to St. Thomas's Hospital the abdomen was very prominent, especially in the epigastric region; numerous dilated veins were seen over the sternum, the blood-stream running upwards. The body was wasted. The liver presented a definite, but not very hard, lower edge; its surface was smooth; there was no pain. A fluid thrill and shifting dullness could be detected in the abdomen. No improvement took place, and the patient was sent to the Lambeth Infirmary in November, 1904.

HISTOLOGY.

The growth is constructed of large polyhedral cells having the usual hepatic characters, closely arranged without stroma, and parted into irregular columns and groups by a fine plexus of intervening capillaries. The cells are aggregated into lobules of varying dimensions, the smallest of which are less in size than the fully-formed lobules of the normal liver. The arterioles and veins are confined to the septa of connective tissue which separate the lobules; there are no proper biliary ducts. Large numbers of the tumour-cells are vacuolated and enlarged, the nucleus of the distended cell being displaced to the periphery and flattened. In sections stained with Sudan III all the usual stages of fatty infiltration so common in hepatic cells are traceable, the fat appearing first in droplets, which eventually coalesce into a single drop, which distends the cell and converts it into a thin-walled sphere. In the smaller groups the component cells may show no such infiltration, and in the fatty lobules themselves there is usually a more or less complete, though narrow, peripheral zone of cells which hold less fat, or may contain none at all.

A certain number of the vacuolated cells contain fine needles of a deep-brown colour. In the strands of connective tissue between the lobules there occur, here and there, collections of pigment of somewhat the same colour, but this is invariably spherular, some of the spherules being notably coarse, and in all respects like the amorphous hæmatoidin resulting from the disintegration of extravasated blood, to which it doubtless owes its origin; and, indeed, extravasated blood-cells occur in connexion with some of these collections. There is no local relationship between the two forms, though small circumscribed collections of spherular pigment may occur between the elements of the tumour, probably in endothelial cells. It is noteworthy, however, that except in the proper cells of the growth, the pigment, however fine, is always spherular ($\frac{1}{2}$ oil immersion). To revert to the acicular crystals within the tumour-cells, it is to be observed that these are found only in connexion with vacuoles, the size of the collection corresponding with that of the vacuole. They are not disposed in tufts or rosettes, but interlace to form a somewhat open latticework. They are more slender than any forms of hæmatoidin (or the identical bilirubin), and I think they must be regarded as bile-stained crystals of fat occupying the intracellular vacuoles. In the case of the lesser vacuoles the crystals fill the whole of the space; in the larger they are confined to the periphery, the more central mass having been dislodged in the preparation of the section.

Let me pass on, however, to the more interesting feature, the amyloid degeneration which the growth presents. In sections stained with methyl violet a widely-spread amyloid disease of the vessels is revealed. Those running in the fibrous septa between the lobules are nearly everywhere affected, their walls being homogeneous, swollen, and of a brilliant deep red colour. The capillaries ramifying between the cells of the growth are affected in very various degrees: in some of the lobules, only one here and there; in others so extensively as to form a plexus, in the meshes of which are contained the cells of the tumour, compressed by the swollen vessels into a finer network than that produced by the

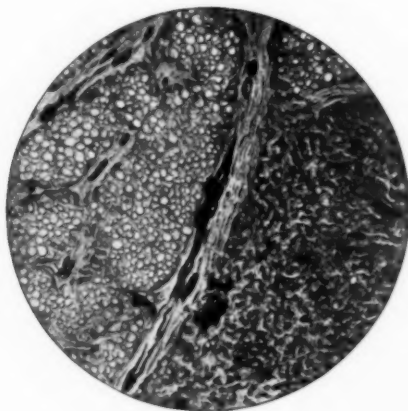


FIGURA 1.

Hepatis adenomatis sectio microscopica degenerationem amyloidem arteriolarum venularumque in telae connexive septis, tumoris inter lobulos, monstrans. Cum oleo cellulae late distenduntur. Vasa affecta quae in sectione rubra tinguntur, in picturâ photographica nigra esse videntur. ($\frac{1}{2}$ obj.)

A microscopic section of the hepatic adenoma described in the text, stained with methyl violet. The distribution of the amyloid disease of the vessels is shown by the darkness of those affected, their red colour appearing as deep black in the photograph. Besides the degeneration of the larger vessels in the connective-tissue septa, a few capillaries in the lobule on the right of the main septum are affected. The cells of the other lobules are vacuolated from the accumulation of fat. ($\frac{1}{2}$ obj.; Oc. 4.)

diseased capillaries themselves. The most affected areas of the growth, in short, exactly resemble the liver itself in advanced amyloid disease, where the change remains confined to the vessels, the hepatic cells merely undergoing the secondary changes due to compression.

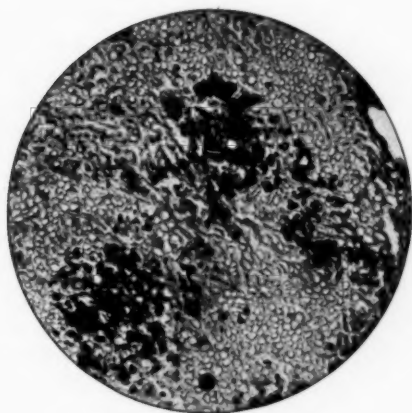


FIGURA 2.

Sectionis ejusdem alia pictura photographica. Monstratur vasorum capillarum plexus inter tumoris cellulas cum degeneratione amyloide affectus. ($\frac{2}{3}$ obj.)

Another portion of the same microscopic section, showing more extensive amyloid disease affecting the capillaries which ramify between the cell-groups. Many of the cells are vacuolated from the accumulation of fat. The net of swollen, amyloid capillaries appears deep black in the photograph. ($\frac{2}{3}$ obj. ; Oc. 4.)

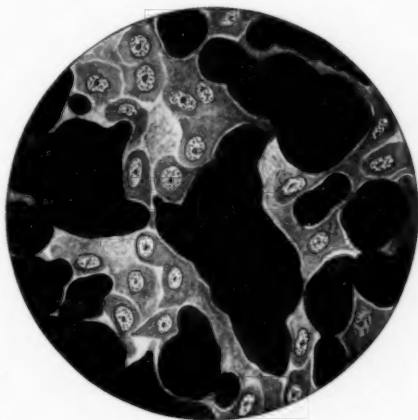


FIGURA 3.

Sectionis ejusdem portio plusquam in figurâ præcedenti magnificata. Monstrantur tumoris cellulae in vasorum capillarum tumidorum rete compressae. ($\frac{1}{2}$ obj.)

A portion of the field shown in the preceding figure, more highly magnified, showing the plexus of swollen amyloid capillaries and the cells of the tumour compressed in its meshes. ($\frac{1}{2}$ obj. ; Oc. 4.)

In the remains of the liver displaced over the growth the typical marks of amyloid disease are obvious to the naked eye. And, as in the tumour, its degree varies in different spots: in some it is confined to small foci of the individual lobules; in others a group of lobules is so diseased as to form an extensive homogeneous, translucent block of tissue. There is no cirrhosis apparent to the naked eye in the unde-generate areas. The amyloid disease of the liver and of the adenoma in it may be attributed to the malaria which the history records the patient to have contracted in India. To the statement made that the patient on one occasion "brought up 6 oz. of tuberculous-looking pus" no significance can be attached, for it was an isolated occurrence, unconnected with any other symptoms of thoracic disease; the material probably came from the stomach.

Of the second of the two tumours, it is enough to say that in its microscopic characters it is identical with the other; it would be difficult to find two things more closely alike, but its vessels present no trace of amyloid disease. The growth exceeds the first in size, being 8 in. (20 cm.) in diameter, of flattened spheroidal form, and for the most part smoothly covered with peritoneum, on the surface of which are the shreddy remnants of adhesions torn through in the removal of the parts. The divided surface is remarkably uniform in structure, and is subdivided into lobules of various sizes by fine septa of connective tissue, though wanting the regularity of size presented by the normal hepatic lobules. Coarser partitions further subdivide the mass into more voluminous lobes. The surface of the section is in many areas of a deep-green colour, which was more brilliant in the recent state, and a proof that the cells of the growth were, in some respects at least, functional. In a few parts the surface is blotched with black, like the section of a pigmented bronchial gland, a feature in which it also resembles the tumour first described; in others there are irregular extravasations of blood, the red colour of which is still obvious.

The patient, a man aged 30, was admitted into St. Thomas's Hospital, under the care of Mr. Makins, to whom I am indebted for leave to publish the case. He had been subject to occasional bilious attacks, associated with vomiting, for the "whole of his life." On March 11, 1908, he was seized with an attack of severe vomiting, accompanied with epigastric pain. The vomiting recurred once or twice in the next week, but his general condition, apart from a subjective feeling of throbbing in the epigastrium, was normal. On admission, there was found a large,

well-defined tumour extending from the costal margin in the central zone to $1\frac{1}{2}$ in. below the umbilicus, and moving with respiration. On April 29 the tumour was exposed and brought out of the abdomen, the pedicle being ligatured and divided, and the growth removed. The patient became very much collapsed, although no large amount of blood was lost, and died during the closure of the abdominal wound. At the autopsy the pedicle of the growth was found to have been attached to the left lobe of the liver. The spleen was somewhat enlarged. There was no further disease.

HISTOLOGY.

The structure is a very fair replica of hepatic tissue, the cells being disposed in lobules separated by connective tissue bearing the larger vessels, and themselves occupying the meshes of a fine capillary network. Most of the cells hold a certain number of fine brownish granules of pigment. Fatty infiltration of the tumour-cells is not to be observed. There are no true ducts—i.e., canals lined with non-granular columnar epithelium—but in the midst of the cell-groups there are many uniformly distributed channels produced by the evacuation of secretion from the cells. The cells bounding such are in no way differentiated from those of the rest of the growth; and the spaces may be regarded as bile canaliculi abnormally distended by secretion in consequence of there being no communication with the proper excretory passages of the liver. They are readily distinguishable from a second plexus, of capillaries, by the absence of endothelial lining. In sections cut from the green areas of the growth, and mounted in glycerine, many of these dilated canaliculi are occupied by homogeneous pale-green material, which may uniformly fill the space or appear as a coarsely spherular aggregate. These plugs present much the same appearance and colour in sections stained with Ehrlich's hæmatoxylin and mounted in xylol balsam after the usual dehydration and clearing. In the sections stained with hæmatoxylin and eosin the material is stained of a pale reddish brown.

Such passages and their green-stained contents were fully and accurately described and figured by Mr. Howard Paul, twenty-five years ago, in the *Transactions of the Pathological Society*.¹ The amount of secretion in the tumours referred to by Mr. Paul was sufficient to give them a bright-green colour: a chromolithograph of one such specimen

¹ *Trans. Path. Soc. Lond.*, 1885, xxxvi, p. 238.

is furnished with the paper. In one of Mr. Paul's four figures, plate ix, the green-coloured growth being an adenoma, the epithelium bounding the channels is columnar; but before the latter can be regarded as true ducts, more detailed information of the intimate structure of the cells would be needed. In the case of the other three figures illustrating the plate, the channels exactly correspond with those shown in the adenoma under notice, though the tumours themselves are all three classed as carcinomata, from their infiltrating character and the

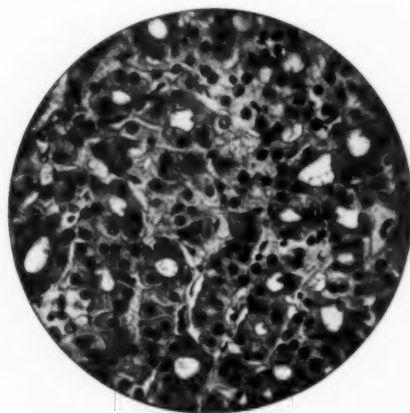


FIGURA 4.

Hepatis adenomatis ingentis (20 cm) sectio. Vasorum capillarium degeneratio abest. In structurâ tumor, opere ablatu, præcedentem simulat. Inter cellulas ipsas, autem, monstrantur ductus qui canaliculi biliares forsan repræsentant. A vasis capillaribus distinguuntur endothelio absente. De his canaliculis quidam substantiam biliverdino tinctam continuerunt. (½ obj.)

A microscopic section of the second hepatic adenoma described, showing its close resemblance to the normal tissue of the liver. The cells lie in the meshes of a fine capillary plexus. Besides the capillaries, the section shows many duct-like passages. These are distinguished from the smaller and collapsed capillaries by the absence of endothelial lining; they are bounded by granular cells like those of the rest of the growth, and represent extra-cellular bile canaliculi, abnormally distended in consequence of their having no communication with excretory passages. (½ obj.; Oc. 4.)

invasion of the portal vessels. Such a dilatation of extracellular bile canaliculi would occur indifferently in either form of growth, seeing that the cells are of hepatic origin and retain to some extent their bile-producing capacity. The material in question is named by the author

cited inspissated bile. Although stained with biliverdin, it owes its solidity, of course, to coagulation artificially produced by the fixing reagents used—to the coagulation of the biliary nucleo-albumin. It need hardly be pointed out that carcinomata arising from the proper bile-ducts fall into a different histological and physiological category; as in the corresponding case of the breast, they would be duct- or columnar-celled growths. To Mr. Paul's account, however, I may add a further detail in the case of the adenoma under notice. And it is that in some of the cells themselves, coarse spherules of similar homogeneous green-stained material are to be met with: I have seen such in the cells actually bounding particular channels which held the same substance. The spherules vary in size, they are distinctly green, and their want of the proper spherical form indicates that they are not droplets of fat.

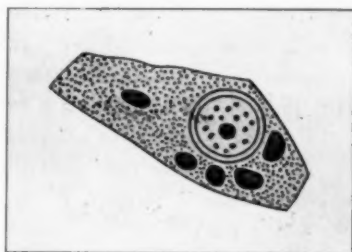


FIGURA 5.

Hepatis tumoris in figurâ 4, repræsentati cellula una. In cytoplasmate monstrantur spherulæ quaedam substantiæ cum biliverdino tinctæ, in canaliculis intracellularibus distentis, sive cystibus intracellularibus, forsitan contentæ. ($\frac{1}{2}$ obj.)

A cell of the hepatic adenoma (Case 2) described, from an unstained section made from material fixed in formalin, followed by alcohol, and mounted in glycerine. In the cytoplasm are five spheroidal blocks of homogeneous, pale-green material, which, from their resemblance to that in the bile passages, may be regarded as of the same nature. The material in question is probably located in the intracellular bile canaliculi, which have become dilated so as to form intracellular retention-cysts. ($\frac{1}{2}$ obj.; Oc. 4.)

These intracellular collections are presumably secretion pent up in abnormally distended intracellular bile canaliculi—in intracellular retention-cysts. That the intracellular substance is not bile-stained glycogen appears from its failing to stain with iodine solution, for after fixation of hepatic tissue, either in formol or alcohol, this substance takes

the same deep-brown colour that it does in the recent state. The black pigmented areas already referred to in the tumour correspond with extravasations of blood in the interlobular connective tissue, the pigment being of a deep orange brown, and free red blood-cells being still recognizable in association with it.

That the cells of a carcinoma do not necessarily lose their physiological capacities is now so well established (though the contrary was once regularly taught) as to render a discussion upon this topic unnecessary. Let me refer only to one of the clearest examples, where both the adenomatous and carcinomatous forms of new growth are met with, as in the case of the hepatic tumours which have just been considered. In the thyroid, then, there are the encapsulated, enucleable adenomata growing within the gland; and there is the adenoid carcinoma which is followed by metastases in the internal viscera and in the bones. In both forms of neoplasm the vesicular structure of the normal thyroid may be accurately reproduced; and in both, the cells may secrete a homogeneous colloid which fills the spaces and completes the normal histological picture. And that the metastatic growths in such circumstances are actually capable of performing the thyroid function is shown by the remarkable case recorded by von Eiselberg in his article in von Bergmann's "Surgery."¹ In this case, after total excision of a thyroid which proved to be "adenomatous," symptoms of cachexia strumipriva supervened; but these abated with the growth of a tumour in the sternum. Six years later this metastasis was removed on account of the pressure symptoms which it produced; it proved to be a "cylindrical-celled carcinoma," secreting colloid. Death occurred from post-operative thyroid tetany.

Some years ago (1895) I put to myself the question whether the cells of a mammary carcinoma performed in any degree the physiological function of mammary epithelium; whether the fat found in certain of the cells of the tumour, and commonly regarded as evidence of a simple degeneration, might not really be the result of a physiological process of the same kind as that which occurs during lactation. The actual concurrence of pregnancy would not be absolutely necessary for such a result. The secretion from the breasts of new-born children, for example, contains the various constituents of milk; and in bitches the secretion of milk, apart from pregnancy, is a well-established

¹ von Bergmann, von Bruns, and von Mikulicz, "System of Practical Surgery" (transl.), 1905, ii, p. 349.

phenomenon. In sections of typical scirrhus carcinomata of the breast stained with Scharlach, extensive groups of the tumour-cells may be found loaded with fine droplets of fat. This one may see in areas in which no traces of softening or other change exist; the cytoplasm is strewn with fat, the nucleus is intact. The presence of fat is, of course, unrecognizable and would be missed, in sections dehydrated in the usual way with absolute alcohol.

The cores of material which often lie in the centre of the larger epithelial columns, in a mammary carcinoma, consist of necrotic epithelium, and as told, in sections stained with Scharlach, they may contain a certain amount of fat. Seeing that the cells of other tumours, however, may exhibit similar fatty changes, the hypothesis put would be difficult to substantiate by so simple an observation. A diagnosis of mammary secretion cannot be founded upon the mere presence of fat; unless, as Mr. Mavrogordato has reminded me, it could be shown by the examination of a sufficient amount of the fat, that butyric acid was extractable in quantity in addition to the commoner forms of oleic and stearic acids.

Johannes Müller, in his work on Cancer,¹ in dealing with the chemistry of carcinoma, has observations which were at that time taken to demonstrate the presence of casein in two examples of carcinoma of the breast. But as the same reaction was obtained in the extract of a "medullary fungus" of the kidney, and in a "pultaceous alveolar cancer" of the skull, the value of the observation almost vanishes. In fact the main test then relied upon is not at the present time admissible. It consisted in boiling the material and finding that acetic acid produced a turbidity in the extract which disappeared on further addition of the acid. Although caseinogen is not coagulated by heat, it has a parallel in nucleo-albumin, which likewise resembles it in being precipitable by acetic acid, and soluble in excess of the latter. And doubtless this was the substance present in the extract of the renal and cranial tumours referred to, and which at that date had not been differentiated. In 1895 I myself, at that time unaware of Müller's statements, tested the hypothesis in a different manner—viz., by seeing whether a clot of casein could be obtained from an unboiled extract of mammary carcinoma by means of rennet. The result in some instances was negative, in others positive. It will be enough if I give the details of the most striking result, using the notes made at the time, and which have not hitherto been published.

¹ "The Nature and Structural Characteristics of Cancer" (Dr. Charles West's transl.), Lond., 1840.

May 25, 1895: Scirrhus of the breast, removed and examined the same day; the tumour, the size of a walnut; the cut surface presented a close series of spaces from which a yellow or pale brownish, creamy-looking material could be expressed; some of the spaces were as much as 2 mm. in diameter. The tumour was minced, pounded in a mortar with a little calcium phosphate and water, and the pulp pressed on wire gauze; the fluid which came through was thick and yellow, holding a considerable amount of fat (some of it doubtless from the fat-cells entangled in the growth); reaction, faintly alkaline. Of the rennet solution (Baird and Tatlock), which was just alkaline, about two volumes were added and mixed, the capsule being then placed in the incubator at 35° C. Six hours later there were thin patches of transparent clot at the bottom of the vessel, which was much increased an hour later, when there were considerable transparent flakes loosely adhering to the bottom. The capsule was kept overnight in a cool place, and the next day the clot was washed in water and preserved in formol solution; it then took the form of small flocculi. The patient was 59 years of age and had been confined four times, the last occasion being twenty-nine years ago. The tumour was first noticed eighteen months before operation; its growth had been more rapid during the last six months.

What might happen in a mammary carcinoma during pregnancy and lactation, is a matter that has not yet been inquired into with this object, either in the case of man; or of the mouse when the subject of spontaneous carcinoma of the mamma.

The Effect of the Injection of the various Tuberculin and of Tubercle Endotoxin on the Opsonizing Action of the Serum of Healthy Rabbits.¹

By R. TANNER HEWLETT.

IN a former paper² I detailed the results of the injection of certain bacterial endotoxins and of tuberculin T.R. on the opsonizing action of the serum of healthy rabbits. It was found that while tubercle endotoxin obtained by the Macfadyen process³ produced a considerable opsonizing effect, tuberculin T.R., in a dose of 0.002 mg., exerted practically no action. The object of the present work was to ascertain if the different tuberculins in varying doses exerted an opsonizing effect on healthy rabbits, and to compare their actions (if any) with the action of tubercle endotoxin.

The tuberculins were obtained from Meister, Lucius and Brüning, of Hoechst, and the dates of preparation on the bottles were as follows: (1) Old tuberculin, May 28, 1909; (2) Tuberculin T.R., September 9, 1909; (3) Tuberculin bacillary emulsion, September 28, 1909. The tubercle endotoxin was prepared on September 21, 1909, by the trituration of a culture of human bacilli, and filtration and subsequent standardization by the estimation of the amount of dry material in a given volume of the solution.

The rabbits were all large, healthy animals, and blood was obtained in Wright's capsules from an ear vein. In all instances, the blood used as the control was taken at the same time as the samples from the inoculated animals, and the specimens for counting the number of tubercle bacilli ingested by the polymorphonuclear leucocytes were made in the usual manner within two or three hours after bleeding the animals. The leucocytes employed were *human* leucocytes, as rabbits' leucocytes were found to be less satisfactory for making the stained films, and the counts were made on fifty cells. All the inoculations of endotoxin and tuberculin were made subcutaneously in the back. The same control animal was used throughout.

¹ Read at the laboratory meeting of the Section at King's College, March 1.

² *Proc. Roy. Soc. Lond.*, B. lxxxi, 1909, p. 325.

³ See "The Cell as the Unit of Life," 1908, p. 274.

In the first series of experiments the following doses were given;—

(1) Old tuberculin: Rabbit II, 0·001 c.c.; Rabbit III, 0·01 c.c.; Rabbit IV, 0·1 c.c.

(2) Tuberculin T.R.: Rabbit V, 0·0005 c.c. (= 0·0001 mg. solid); Rabbit VI, 0·005 c.c. (= 0·001 mg. solid); Rabbit VII, 0·05 c.c. (= 0·01 mg. solid).

(3) Tuberculin bacillary emulsion: Rabbit VIII, 0·0005 mg.; Rabbit IX, 0·005 mg.

(4) Tubercle endotoxin: Rabbit X, 0·0005 mg.; Rabbit XI, 0·005 mg.

The opsonic indices of all the rabbits were estimated just previous to inoculation, being compared with the control, Rabbit I., and at varying periods after inoculation. The results are summarized in Table I.

From Table I, it will be seen that the maximum variation of the opsonic index before inoculation was between 0·8 (Rabbit II) and 1·28 (Rabbit VIII). The old tuberculin produced little effect; the effect of the two smaller doses (0·001 c.c. and 0·01 c.c.) was practically *nil*, and the largest dose (0·1 c.c.), beyond causing after twenty-fours a negative phase, the index dropping from 1·1 to 0·7, also produced practically no effect. Tuberculin T.R. similarly produced little or no effect beyond the limits of experimental error. Tuberculin bacillary emulsion: The smaller dose (0·0005 mg.) produced little effect, the larger dose (0·005 mg.) caused some rise in the opsonic index, from 0·8 to 1·45 (Rabbit IX) on the eighth day after inoculation. Tubercle endotoxin: The smaller dose (0·0005 mg.) produced apparently some rise in the opsonic index, from 1·06 to 1·4 (Rabbit X), on the thirteenth day after inoculation. The larger dose (0·005 mg.) produced a negative phase, the index dropping from 1·2 to 0·72 (Rabbit XI) on the second day after inoculation. When the experiment terminated the index appeared to be rising, and it was unfortunate that further observations were not made.

In view of the comparatively small effect produced by the inoculations, second inoculations with larger amounts were given on October 19 (fourteen days after the first inoculation) to some of the animals, viz.:—

(1) Tuberculin, T.R.: 0·1 c.c. (= 0·2 mg.) was given to Rabbit V.

(2) Tuberculin bacillary emulsion: 0·05 mg. was given to Rabbit VIII.

(3) Tuberculin endotoxin: 0·05 mg. was given to Rabbit X.

Since the doses, varying from 0·001 c.c. to 0·1 c.c., of the old tuberculin had produced so little effect, it was not thought that any useful purpose would be served by trying an increased dose. The results are summarized in Table II.

TABLE I. (FIRST INOCULATION, OCTOBER 5, 1900.)

Rabbit	October 5*		October 6†		October 7		October 8		October 11		October 13		October 18	
	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index
(I) Control ...	125	1.0	91	1.0	106	1.0	95	1.0	63	1.0	88	1.0	86	1.0
(II) 0.001 c.c. old tuberculin ...	109	0.87	88	0.96	109	1.0	90	0.95	69	1.0	79	0.9	88	1.0
(III) 0.01 c.c. old tuberculin ...	130	1.04	72	0.8	109	1.0	100	1.05	76	1.1	68	0.7	79	0.93
(IV) 0.1 c.c. old tuberculin ...	135	1.1	64	0.7	98	0.92	99	1.05	73	1.08	91	1.08	92	1.07
(V) 0.0005 c.c. tuberculin T.R.	146	1.16	80	0.86	103	1.0	110	1.16	71	1.08	98	1.1	96	1.1
(VI) 0.005 c.c. tuberculin T.R.	121	0.93	72	0.8	87	0.82	88	0.95	77	1.1	106	1.17	99	1.15
(VII) 0.05 c.c. tuberculin T.R.	152	1.2	79	0.86	113	1.07	105	1.1	73	1.08	101	1.12	101	1.17
(VIII) 0.0005 mg. bacillary emulsion	160	1.28	100	1.1	108	1.0	101	1.05	69	1.0	97	1.11	98	1.14
(IX) 0.005 mg. bacillary emulsion	101	0.8	88	0.96	83	0.8	120	1.26	75	1.1	128	1.45	102	1.2
(X) 0.0005 mg. tubercle endotoxin	133	1.06	82	0.9	118	1.1	118	1.26	81	1.2	104	1.17	119	1.4
(XI) 0.005 mg. tubercle endotoxin	150	1.2	78	0.86	76	0.72	107	1.17	84	1.2	99	1.2	110	1.3

* Before inoculation. † Twenty-four hours. The counts were made on fifty cells.

TABLE II. (SECOND INOCULATION, OCTOBER 19, 1900.)

Rabbit	Original Index	October 20*		October 21		October 22		October 26		October 29		November 2		November 12		December 1		December 13	
		No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index	No. of T.B.	Index
(I) Control ...	1.0	78	1.0	111	1.0	117	1.0	96	1.0	108	1.0	98	1.0	93	1.0	101	1.0	155	1.0
(V) 0.1 c.c. tuberculin T.R.	1.16	68	0.87	121	1.1	125	1.07	120	1.25	141	1.3	130	1.2	98	1.05	98	1.0	158	1.0
(VII) 0.05 mg. bacillary emulsion	1.28	75	1.0	110	1.0	132	1.04	135	1.4	155	1.4	131	1.3	90	1.0	115	1.1	155	1.0
(X) 0.05 mg. tubercle endotoxin	1.06	69	0.9	158	1.4	148	1.26	175	1.8	170	1.57	152	1.55	+	—	119	1.2	173	1.1

* Twenty-four hours after second inoculation. † Specimen spoilt. The counts were made on fifty cells.

From Table II, it will be seen that the large dose of tuberculin T.R. caused a slight negative phase, the index dropping from 1.1 to 0.87 (Rabbit V) the day after inoculation, followed by a rise to a maximum of 1.3 ten days after inoculation. The large dose of bacillary emulsion produced apparently a slight negative phase, followed by a rise to 1.4 (from an initial 1.14) on the seventh and ninth days after inoculation (Rabbit VIII). The large dose of tubercle endotoxin produced a pronounced negative phase, the index dropping from 1.4 to 0.9 (Rabbit X) the day after inoculation, the index subsequently rising to a maximum of 1.8 on the seventh day after inoculation.

CONCLUSIONS.

(1) The old tuberculin produces practically no effect on the opsonizing action of the serum of healthy rabbits.

(2) On the whole, tubercle endotoxin seems to produce a more marked effect on the opsonizing action of the serum of healthy rabbits than either tuberculin T.R. or tuberculin bacillary emulsion.

(3) This suggests that tubercle endotoxin might be a more active therapeutic agent than either of the three tuberculins—old tuberculin, tuberculin T.R. and tuberculin bacillary emulsion.

I have to express my best thanks to Mr. Wellcome for the facilities he has afforded me at the Wellcome Physiological Research Laboratories for carrying out this work, and to Mr. E. Thompson, laboratory assistant, on whom the labour of making the counts of the opsonic determinations has fallen.

A Lipoclastic accelerating Action of Serum as an Index in Pathological Conditions.¹

By J. A. SHAW-MACKENZIE and O. ROSENHEIM.²

INVESTIGATING the lipolytic (preferably termed the lipoclastic) activity of serum, we were able to confirm previous statements on the subject, that a true fat-splitting enzyme does not occur in serum, whilst even the action of what might be called a butyrase on the esters of lower fatty

¹ Read at the laboratory meeting of the Section at King's College, March 1.

² From the Physiological Laboratory, King's College, London.

acids is a feeble one. During the course of this work, however, we made the observation that serum, although not possessing any fat-splitting action by itself, nevertheless increases in a remarkable way the activity of pancreatic lipase contained in pancreatic extracts as well as in pancreatic secretion. This property is possessed to a greater or less degree by all the animal sera which we have examined—namely, horse, ox, pig, dog, cat, rat, mouse, rabbit, marmoset. This accelerating action of serum is not specific, in so far as the lipolysis by pig's lipase is increased in the same way by the addition of pig's serum, as well as by that of other animals, whilst, on the other hand, the lipase of the ox is acted upon also by the serum of the pig.

On examination of normal human serum we found that its accelerating power, as compared with the same quantity of animal serum, is about half of the latter. In certain pathological conditions, however, this reaction is increased to double, and even four times, that in health.

[In addition to serum we also examined the cerebrospinal fluid of the dog and cerebrospinal fluid from cases of general paralysis, which were found to be practically inactive. Serous effusions, so far examined, have shown the reaction to a marked degree. We examined also a series of press juices of various animal organs, and found (in the absence of a lipoclastic action of the juice itself) an accelerating action in the case of the testicle and thyroid, whilst in the case of the duodenal press juice the result corresponded to the sum of the lipoclastic action of pancreas and duodenum. In the case of glycerine extracts, those of the testicle, thyroid, ovary, and spleen were found to be slightly active, whilst no acceleration was observed in the case of glycerine extracts of lymphatic glands and prostate.]

With regard to the chemical nature of this accelerating substance in serum very little can be said at present. It withstands the temperature of boiling water; it is not destroyed by putrefaction; it dialyses and is soluble in dilute alcohol. An important property of the accelerator was brought to light by the investigation of its behaviour towards cholesterin, which in all cases was found to exert an inhibitory action.

Details of the method of obtaining a pancreatic extract rich in fat-splitting properties, the various substances which accelerate this action still further, together with the inhibitory action of cholesterin on all of these, have been communicated elsewhere and will be published shortly. Briefly, however, it may be stated that the lipoclastic action is estimated by the amount of decinormal potash used for the neutralization of the fatty acids set free by pancreatic lipase acting on olive-oil emulsion, or

on ethyl butyrate, the mixture being incubated at a temperature of 37°C. for a certain time.

By adjusting the conditions of the experiments in such a way as to reduce the amount of pancreatic lipase and olive-oil emulsion (or of ethyl butyrate) to a minimum, we have been able to show the accelerating power of as little as 0.1 c.c. of serum. Generally, 0.25 c.c. and 0.5 c.c. of serum are sufficient. In this way one is able to compare the degree of acceleration of serum in health and disease. As a matter of fact our first observation on this accelerating reaction was made in a case of carcinoma.

Taking normal human serum as a basis of comparison, the following figures give the results in a typical case: 0.25 c.c. of glycerine extract of pancreas, 0.5 c.c. of water and 2.5 c.c. of olive-oil emulsion were mixed, and after incubation the amount of acid liberated required 5.3 c.c. of decinormal potash to neutralize it. When 0.1 c.c. of serum had been added, this figure rose to 7.4; when 0.25 c.c. serum was added, it increased to 8; and when 0.5 c.c. serum was added, it rose to 9.7. Or in tabular form:—

TABLE I—AVERAGE NORMAL SERUM.

	c.c.	$\frac{N}{10}$ KOH	Acceleration
Pancreatic extract alone	5.3	—
Addition of serum, 0.1 c.c.	7.4	+ 2.1
" " 0.25 c.c.	8.0	+ 2.7
" " 0.5 c.c.	9.7	+ 4.4

The following tables summarise our results when we investigated in a similar way the action of serum and serous effusions from pathological cases. Table II gives results with serum from cases of carcinoma, Table III those with serum from non-malignant cases, Table IV those with serous effusions in carcinomatous and non-malignant cases. The first of these was cedema fluid from the arm, and the remainder ascitic fluids. In these cases the amount of fluid available was larger than with serum, and the figures are proportionately higher.

TABLE II—SERUM IN CARCINOMA.

Case	Serum added	Acceleration
Recurrent carcinoma; breast ...	0.25 c.c. ...	+ 13.7
Carcinoma of rectum ...	0.25 c.c. ...	+ 7.5
	0.5 c.c. ...	+ 11.8
Carcinoma of stomach ...	0.25 c.c. ...	+ 4.9
	0.5 c.c. ...	+ 10.6
Recurrent carcinoma; breast ...	0.25 c.c. ...	+ 5.2
	0.5 c.c. ...	+ 9.2
Recurrence of carcinoma of neck ...	0.07 c.c. ...	+ 7.2

TABLE III—SERUM IN NON-MALIGNANT DISEASE.

Case	Serum added	Acceleration
Syphilis, under treatment, one year ...	{ 0.25 c.c. ...	+ 4.6
	{ 0.5 c.c. ...	+ 5.9
	{ 0.1 c.c. ...	+ 5.6
Glycosuria	{ 0.25 c.c. ...	+ 8.1
	{ 0.5 c.c. ...	+ 10.6
Tabes	0.5 c.c. ...	+ 16.2

TABLE IV—SEROUS EFFUSIONS (CARCINOMA AND NON-MALIGNANT).

Case	Fluid added	Acceleration
Edema of arm, secondary to recurrent carcinoma of breast	2.5 c.c. ...	+ 12.3
Ovarian, malignant tumour	2.5 c.c. ...	+ 33.0
Peritoneal carcinoma	5.0 c.c. ...	+ 27.8
Ovarian, malignant tumour	5.0 c.c. ...	+ 26.8
Cirrhosis, liver (non-malignant)	5.0 c.c. ...	+ 34.1
Cirrhosis of liver ? (non-malignant)	5.0 c.c. ...	+ 30.1

It may be mentioned also that in some of the sera as used for therapeutic purposes a lipoclastic acceleration was observed. For example, 5 c.c. of added antidiphtheritic horse serum gave an acceleration of + 14.6, and 5 c.c. of normal horse serum + 27.1.

It will therefore be seen from the experiments that carcinoma is not the only pathological condition in which the lipoclastic acceleration of serum occurs.

Some fifteen months ago Professor Halliburton kindly afforded one of us (J. A. Shaw-Mackenzie) the opportunity of working in the physiological laboratory of King's College on the tryptic or antitryptic power of the blood, more especially in relation to carcinoma. According to experiments performed concurrently with the lipoclastic investigations, it may be stated that the antitryptic power of serum would seem to run parallel with the lipoclastic acceleration, in normal and carcinomatous conditions. But in the non-malignant cases so far examined, which show an increased lipoclastic acceleration, the antitryptic power would seem to be normal. Many more observations, however, appear necessary before one can state definitely that this is a general rule.

We desire to express our thanks to those who have kindly provided us with specimens in this preliminary inquiry—Dr. J. C. Matthews, Dr. R. H. C. Gompertz, Dr. F. W. Mott, Dr. E. L. Holland, Sir A. Fripp, Dr. Vernon Jones, Staff-Surgeon J. R. Muir, R.N., Dr. Porter Parkinson, Dr. W. E. Dixon, Surgeon D. P. Chapman, R.N., Dr. B. Hughes, and Dr. J. D. McCulloch.

Some Points in the Pathology of Acne.¹

By ARTHUR WHITFIELD.

THE fundamental error in acne is seborrhœa. There is at present a lack of knowledge as to the causation of seborrhœa, but it is known that it is common in adolescence, in disorders of the alimentary tract, and is apparently an inborn peculiarity in some individuals. The symptoms of acne vulgaris are, as we may say, built up on seborrhœa as a basis, and comprise, first, the production of a horny plug in the pilo-sebaceous follicle, the so-called blackhead or comedo; secondly, suppuration around the comedo; and, thirdly, the production of persistent granulomatous nodes, or, in some cases, of cyst-abscesses.

The bacteriology of the condition has been studied for many years by several investigators, and the views held by the various workers are divergent. Unna and Hodara found a small bacillus present, which they did not cultivate, and came to the conclusion that it was the cause of the suppuration. Sabouraud also found the same bacillus and cultivated it, and believed that it was the cause of the seborrhœa and the comedo, but not the cause of the suppuration, which he considered was due to the pyogenic staphylococci. Gilchrist believed that the bacillus was the cause of the symptoms of acne, and named the bacillus the *Bacillus acnes*. Some years ago I investigated the question and came to the conclusion that the seborrhœa was not bacterial in origin, that the comedo was caused by the small bacillus, which, following Sabouraud, I shall refer to as the microbacillus, and that the suppuration was caused by *Staphylococcus pyogenes albus*. Recently Fleming worked at the subject and came to the conclusion that the suppuration was in most cases caused by the microbacillus, and urged in proof of his contention its invariable presence in the lesions, its power when rubbed into the arm of producing suppuration in some people, the variable opsonic index in acne patients, and the beneficial effect of inoculation. The appearance of Fleming's work stimulated me to renew my investigations, especially because of the very considerable proportion of acne pustules in which he was unable to demonstrate staphylococci. I therefore examined pus from unopened pustules in my next twenty cases, and in nineteen of these I

¹ Read at the laboratory meeting of the Section at King's College, March 1.

found the cocci in smears, and in the twentieth I was able, by cutting horizontal sections of an acne pustule, to show the cocci in active colony at the periphery of the abscess. My attention was then drawn by Dr. Molesworth, who was working in my clinic, to a paper by Hallé and Civatte in the *Annales de Dermatologie* for 1908. It is worthy of note that this paper was on the bacteriology of the sebaceous glands, and the bacillus described by them and afterwards found to be identical with the microbacillus was found in the little plugs (really minute comedones) of patients who were not suffering from acne. The importance of this paper was the publication of the fact that the microbacillus was an anaerobe, thus obviating all the difficulty previously experienced in getting pure and abundant culture of the organism. I may state that this fact was apparently rediscovered independently in this country last year by Südmersen and Thompson. Dr. Molesworth undertook an investigation of this organism, and my cultural experiments were made subsequently to his. His paper is not yet in print, and it is only by his kindness that I am able to add the cultural details to this paper.

It is well known that vast colonies of this bacillus are found in the mouths of sebaceous glands of patients who are not suffering from obvious acne, but it will be found that in every instance there is present a minute comedo in the mouth of the follicle. The protagonists of the pyogenic effects of the microbacillus meet this objection by saying that the microbacillus is not pyogenic for every individual. It is, however, the fact that in marked acne only a small percentage of the comedones become surrounded by suppuration, while all are stuffed with the bacillus in the most active growth. Therefore the same bacillus must be pyogenic at one point and non-pyogenic at another in the same section not more than a millimetre away. This is to me inconceivable, whereas it is quite simple if the bacillus is considered as the cause of the comedo and the suppuration the result of a secondary infection with another organism. If one examines the bacillus in a smear preparation from a comedo, one finds that it is definitely Gram-positive; whereas if one examines the bacilli found in acne pus, those furthest away from the macerated comedo are found to be much less definitely Gram-positive, and I think it is commonly admitted that with the death of bacilli the quality of staining with Gram becomes progressively lost. That these bacilli are not actually dead is shown by the fact that one can grow them, and that whereas the first generation so grown will be weak Gram-stainers, the second and later generations regain their positive staining with Gram.

As regards the opsonic index, my investigations of a small number of cases gave me rather indefinite results, the lowest being 0.77, the highest 1.03. Dr. Emery carried out a couple of agglutination tests for me, and found that the serum of neither healthy nor acne patients gave any sign of agglutination. This is against Gilchrist's statement that he could identify every acne patient from his serum alone by the agglutination test. Curiously enough, Gilchrist found that the agglutination was more marked in the higher dilutions up to 1 in 100 than in the stronger, a remark to which Dr. Emery drew my attention. Fleming says that his bacillus, if made into a vaccine and injected in larger doses than 10 million, causes marked aggravation of the symptoms. I have given of the microbacillus several doses of 1,000 million, and a great many of doses varying from 250 to 500 million, without any marked aggravation of the symptoms.

I prefer not to say much about the therapeutic effects of this vaccine, though I believe it will prove valuable. I am at present treating a test case under the observation of a dermatological colleague, as I believe we are more likely to get at the truth by having our results controlled by an impartial observer.

Anyone who attempts the cultivation of the organism in 2 per cent. glucose agar under anaerobic conditions will, I think, be convinced of the ease with which the organism can be cultivated, and from the enormous numbers of colonies so obtained will also be convinced that the organism obtained is that which is present in such large numbers in the comedo.

If Fleming's organism is so toxic that 50 million give rise to marked suppuration in an acne patient, I think it cannot be the same organism as Dr. Molesworth's and mine, which gives rise to no special trouble in doses of from 250 to 1,000 millions.

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OTOLOGICAL SECTION.

CONTENTS.

December 4, 1909.

	PAGE
Audible Tinnitus. By HERBERT TILLEY, F.R.C.S.	1
Summary of the Post-operative Tests in Eight Cases of Labyrinthine Disease. By RICHARD LAKE, F.R.C.S., and NORMAN PIKE, M.B.	2
Demonstration of Hays' Pharyngoscope. By W. MILLIGAN, M.D.	9
Lantern-slide Demonstration, illustrating the Pathological Changes found in the Ear of Deaf-mutes. By A. A. GRAY, M.D.	11
Long-continued Suppuration (Aural) with Spontaneous Cure? By G. N. Biggs, M.B.	15

February 5, 1910.

Letter from Professor POLITZER	17
By SYDNEY SCOTT, M.S. :—	
(1) Microscopical Sections through the Mastoid Antrum in a Fatal Case of Scarlet Fever demonstrating Streptococcus Conglomeratus <i>in situ</i>	18
(2) Demonstration of some Anatomical and Pathological Preparations ...	22
Deafness and Discomfort in the Right Ear as Early Symptoms in a Case of Epithelioma originating near the Right Eustachian Tube. By EDWARD LAW, M.D.	28
Endothelioma of the Temporal Bone. By J. DUNDAS GRANT, M.D.	32
Some Features of the Auditory Apparatus of a 16 mm. Human Embryo, as shown in a Reconstruction Model (by the Wax-plate method of Born). By C. J. JENKINS, F.R.C.S.	34
Pulsating Angioma (Angioma racemosa, Cirroid aneurysm) of the Right Auricle. By GEORGE WILKINSON, F.R.C.S.	35

	PAGE
Objective Clicking Sound in Left Ear. By HERBERT TILLEY, F.R.C.S. ...	36
Tabetic Deafness. By DAN MCKENZIE, M.D.	38
An Acoustic Aid for Persons partially Deaf from various Causes. By H. MACNAUGHTON-JONES, M.D.	40

March 5, 1910.

Twenty Specimens of Chronic Middle-ear Suppuration and its Sequela, Eighteen of the Bones being of the Infantile types and two Cellular. By ARTHUR H. CHEATLE, F.R.C.S.	41
By W. MILLIGAN, M.D. :—	
(1) Pedunculated Papilliform Growth, springing from the Posterior Border of the Cartilaginous Meatus	44
(2) Large Nasopharyngeal Growth in a Boy aged 12	47
(3) Demonstration of Kuhn's instruments for Per-oral Intubation	49
By DAN MCKENZIE, M.D. :—	
(1) Case with Well-defined and Transitory Ménière's Symptoms ;	
(?) Migraine with Auditory and Vestibular Phenomena	50
(2) Thickening of the Cartilage of both Auricles (?) Othæmatoma	53
Audible Tinnitus. By ALEXANDER SHARP, F.R.C.S.Ed.	54
Mucous Polypus presenting at the Pharyngeal Orifice of the left Eustachian Tube in a Man suffering from Bilateral Chronic Adhesive Otitis Media. By J. ARNOLD JONES, F.R.C.S.Ed.	55
Osteomyelitis of the Right Temporal Bone, secondary to Mastoid Disease ; Removal ; Recovery. By HUNTER TOD, F.R.C.S.	57
By H. J. DAVIS, M.B. :—	
(1) Adventitious Membrane, resembling Normal Drum, formed after Radical Mastoid Operation	58
(2) (?) Papilloma on the Posterior Superior Quadrant of the Right Drum	60
(3) Left Facial Paralysis following a mild Catarrh of the Middle Ear. No Perforation	60
(?) Congenital Defect of both Tympanic Plates. By C. ERNEST WEST, F.R.C.S.	61
Endothelioma of Temporal Bone. By D. R. PATERSON, M.D.	61

June 4, 1910.

Notes on the Pathogeny of Cholesteatomata. By V. H. WYATT WINGRAVE, M.D.	63
Deformity of both Pinnae resulting from Perichondritis following Double Mastoid Operation. By H. J. DAVIS, M.B.	71
Temporo-sphenoidal Abscess in which Vomiting was entirely Absent. By A. L. WHITEHEAD, B.S.	73

Contents

V

	PAGE
Hysterical Deafness with Active Vestibular Reactions. By P. H. ABERCROMBIE, M.D., and DAN MCKENZIE, M.D.	74
By HUNTER TOD, F.R.C.S.:—	
(1) Exostosis of Right External Meatus in a Boy aged 10	76
(2) A Case in which the Clinical Symptoms simulated a Cerebellar Abscess. Brain explored on two occasions. No Abscess discovered. Recovery	79
(3) Specimen of Cholesteatoma or Keratosis Obturans of the External Auditory Canal	81
Two Cases of Cholesteatoma of Unusual Size extending into the Posterior Fossa and Causing Obliteration of the Sigmoid Sinus. By GEORGE WILKINSON, F.R.C.S.	82
Malignant Growth of the Right Temporal Bone, with Extension through the External Meatus, resembling an Aural Polypus. By P. WATSON WILLIAMS, M.D.	85
Specimen of Fracture of the Temporal Bone. By G. J. JENKINS, F.R.C.S. ...	88

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Otological Section.

December 4, 1909.

Dr. EDWARD LAW, President of the Section, in the Chair.

A Case of Audible Tinnitus.

By HERBERT TILLEY, F.R.C.S.

MISS G., aged 13, sought advice for a "purring" noise in her right ear, which she had noticed for at least two years, but which had become louder during the past twelve months after an attack of "influenza." Hearing in both ears is normal, and no pathological conditions are to be seen in the meatus nor in the nasopharynx. The general health is excellent. She has menstruated once—about four months ago—but this seems to have exerted no influence for good or evil upon the aural symptom. Listening through the otoscope, it may be noted that : (1) The "purring" sound—very like a hæmic murmur—is best heard when the head is erect, and it is synchronous with the pulse. (2) It is diminished by either very light pressure over the right carotid artery, or by much firmer pressure on the left. Tilting the head towards the left shoulder has also a similar effect. The pulsating murmur can be heard in a quiet room if the observer's ear be placed close to the patient's right ear, and is just perceptible if a stethoscope be applied to the temporal bone above the pinna.

DISCUSSION.

Mr. CRESSWELL BABER said he showed a similar case some years ago in which there was a purring noise in a young woman. It was, he thought, associated with anæmia.

Dr. H. J. DAVIS thought that if she were given five drops of perchloride of iron with $\frac{1}{2}$ dr. of dilute hydrobromic acid t.d.s. the noise would be much

2 Lake & Pike: *Post-operative Tests in Labyrinthine Disease*

diminished. The murmur was a rough and scratching one, somewhat like the murmur heard over the eyes in exophthalmic goitre.

Mr. WAGGETT said it would be an interesting experiment to tie the external jugular vein. If this vein were pinched without deep pressure, the murmur at once ceased. He had a somewhat similar case in an old lady, who had an audible murmur for half an hour after eating.

Mr. C. G. LEE said that some years ago he saw a young lady who had been trying to emulate her brother in carrying a sack of flour across the room. She did so, but immediately after she heard a noise in her ear. She consulted him within a week, and the bruit was then easily audible. She was now married and had a family, so he supposed the aneurysm, which he presumed it was, had disappeared.

Dr. FITZGERALD POWELL suggested that before any vigorous treatment was adopted efforts should be made to raise the blood pressure. He thought 10 m or 15 m of adrenalin should be given three times a day.

The PRESIDENT (Dr. Law) said the noise was stopped by the slightest pressure on the vessels of the affected side, and considerably lessened by stronger pressure on the vessels of the opposite side of the neck.

Mr. A. L. WHITEHEAD doubted the necessity of doing anything, as the child did not suffer any discomfort. Compression of the external jugular did not altogether stop the tinnitus; therefore ligature of this vessel would only give partial relief, which would probably not satisfy the patient.

Mr. HERBERT TILLEY, in reply, said it was difficult to know how long the noise had been present; the mother that day said it must have been there two years ago, as the child "thought there was a bee in the room." He did not believe it was constitutional, or that iron or adrenalin would act beneficially in any way. If it were constitutional, probably a similar murmur would be heard over the jugular vein or pulmonary artery. She had had no illness, and there were no cardiac or circulatory lesions. He agreed with Mr. Whitehead as to operation: no operation should be undertaken unless the noise became a nuisance to her, in which case it might be well to tie the external jugular.

Summary of the Post-operative Tests in Eight Cases of Labyrinthine Disease.

By RICHARD LAKE, F.R.C.S., and NORMAN PIKE, M.B.

THE notes upon the histories of the cases are only intended to present the reader with a general view of the course of events; the points we wish to place before the Section being the results of the tests for labyrinthine disease after operation, mastoid or labyrinthine.

Case I.—J. H., male, aged 35. First attendance, September 12, 1907: Discharge from right ear, which had been intermittent for six or seven years. Deaf with right ear as long as patient could remember; worse lately. Humming tinnitus in right ear. A few weeks after first visit, an attack of vertigo; confined to bed three days; objects moved up and down. August, 1907: Severe attack of vertigo after running for train; duration, four minutes. Operation, September 25, 1907: Stenosis of external meatus; perforation of Shrapnell's membrane; depression seen in the bone where prominence of canal should be. Subsequent history: No vertigo since operation. Present state: Thinks he hears better than before operation. Tests: Caloric. Right ear, normal. Rotatory, 10 turns to right nystagmus, 25 seconds; no vertigo. Rotatory, 10 turns to left nystagmus, 25 seconds; slight vertigo.

Case II.—J. E., male, aged 55. Frequent attacks of vertigo for three or four years. Suppurative otitis of long duration in left ear. First operation, 1904: Left radical mastoid; the bone over external semicircular canal destroyed, and membranous canal exposed for about $\frac{3}{16}$ in., which was removed. Subsequent history: Three and a half years almost free from vertigo, then severe attack lasting thirty-six hours, with vomiting and nausea. Second operation: Ablation of labyrinth, February, 1908; vestibule opened up with chisel and gouge, interior swabbed with 40 per cent. formalin; foramen ovale opened and good passage made into vestibule. After operation, nystagmus on rotation. Present condition: Since second operation no vertigo, no air conduction to tuning-fork 16 C to C₂₀₄₈. Tests: Caloric. Left ear, no reaction; right ear, normal; no spontaneous nystagmus.

Case III.—G. F., male. Discharge from left ear nearly all his life; tinnitus in the right for some years; occasional attacks of vertigo, which became worse and of greater frequency of late, occurring almost daily; staggered chiefly to the left side, objects moved horizontally, had to stay in bed for eight weeks; nystagmus more marked during the attacks. Operation, May, 1908: Left radical mastoid. Sinus found leading into left external canal. Tests: Acoumeter, 0; voice, 4 in.; Rinné negative; bone conduction slightly diminished. Subsequent history: Ear dry—much better—though complained of swaying when standing. Feeling of pressure on top of head, no tinnitus, no spontaneous nystagmus; on turning head sharply to the left, nystagmus > on looking to right of short duration; unable to work, and complained constantly of inability to work; caloric test, left strong. Second operation: Vestibule entered through external and posterior canals; facial nerve exposed. After the

4 Lake & Pike: *Post-operative Tests in Labyrinthine Disease*

operation, slight nystagmus on looking to the left of only one week's duration. No vertigo, facial paresis incomplete on third day (now well), patient up upon fifth day. Present state: No spontaneous nystagmus. Tests: Caloric. Left negative; rotatory 10 turns to right; nystagmus 3 seconds; rotatory 10 turns to left; nystagmus 18 seconds.

Case IV.—F. A., male. Discharge from right ear, twenty-seven years; cephalalgia, two years; vertigo, three attacks last week; falls to left. Operation, June, 1907: Radical mastoid. Prominence of external canal seen to have been destroyed. Subsequent history: On syringing, head falls to right; right pupil dilated. July 1—Severe vertigo, falls to right; July, 1907—Labyrinth opened. Post-operative nystagmus greatest excursion to right. Present state: No spontaneous nystagmus; rotatory test 10 by right, 20 seconds, very slight vertigo; 10 by left, 18 seconds, rather more vertigo. Caloric. Right negative; left rapidly produced vertigo; patient falling to right (nystagmus not able to be noted).

Case V.—Nurse B., female. Past history: Discharge from left ear since childhood; frequent attacks of vertigo. Operation, March, 1908: Radical mastoid. Returned to work June 11, wound behind ear not healed. Later, discharge from ear, occasional cephalalgia and vertigo, slight nystagmus. Symptoms point to labyrinthine or intra-cranial lesion. Operation, December 10, 1908: Labyrinthine operation. No pus in labyrinth; escape of cerebrospinal fluid; recovery tedious; dressings often soaked in cerebrospinal fluid; pyrexia, seven days; marked lateral nystagmus; complains of headache and giddiness; tendency to lie on right side; became very ill; incontinence of urine and wandering. December 30: Lumbar puncture; clear fluid; O.D. a little blurred; some pus in depth of wound. January 6, 1909: Nystagmus more marked on looking to left (according to Neumann and Bárány this points to an intracranial lesion). Giddy when she sits up; tendency to fall to right. Left, January, 1909: Slight spontaneous nystagmus.

Case VI.—H. S., male. Past history: One year vertigo; old otitis media suppuration; perforation in Shrapnell's membrane; pressure on external meatus caused vertigo (hearing good). Operation, January 22, 1908: Removal of left labyrinth. Left external canal and vestibule opened, &c. (extensive destruction of external canal found). Subsequent history: Slight vertigo after walking. Present state: Slight spontaneous nystagmus, right and left; caloric test, cold, left negative; right normal, with some vertigo.

Case VII.—A. M., aged 47, male. Past history: Left ear has discharged for many years; refused by life-assurance company. Operation, October 16, 1907: Radical mastoid, left. Stayed in bed after operation ten days; on getting up was weak and could not stand alone, not feeling steady; this feeling of unsteadiness lasted another six days; objects did not move. Subsequent history: Never had a definite attack of vertigo, but if he turns the head suddenly in any way, he feels unsteady for a short time. Present condition: No spontaneous nystagmus. Caloric reaction, cold, left ear negative, right slight, but definite reaction. Rotatory test, 10 by left, 19 seconds, very slight vertigo; 10 by right, 7 seconds; left ear dry; voice heard in concha; Rinne negative; bone conduction diminished; with eyes shut and feet together tends to fall backward; walks backward with eyes shut fairly well.

Case VIII.—V. D., female, aged 22. Had polypus removed from right ear in childhood; ear was dry, and gave no trouble until January, 1908, when free discharge began and continued; six weeks ago facial paralysis developed with mastoid tenderness; no vertigo. Confined, June, 1908. Present state: There is extreme tenderness over mastoid (right) extending for some distance; meatus externus choked with polypi. Operation, July 1, 1908: Radical mastoid. Lateral sinus situated anteriorly and superficially. Tests before operation: Conversation voice, 3 in. Present state: Conversation voice, 1 yd. Caloric reaction right negative. Rotatory test, 10 by right, strong nystagmus, 25 seconds; 10 by left, very slight nystagmus.

The cases we are presenting to the Section have not been selected in any way to illustrate peculiar points, but consist of those cases which have been attending at the hospital during the time we have been working together, and they are brought forward in the hope that they will prove of interest as having a bearing on still moot points, and as being in general of interest from the point of view of labyrinthine symptomatology. The points to which we would draw attention are that all the cases, with the exception of one, suffered from vertigo during recovery from the radical mastoid operation, although this was in every instance brought to a completely successful issue; that post-operative vertigo continued in three cases, and was so severe as to necessitate destruction of the labyrinth; that in two of the cases, Nos. VII and VIII, although vertigo was absent in Case VII, and there was no history of a definite attack, but only of some slight loss of equilibrium, yet in both these cases the caloric irritability on the same side as the radical mastoid was negative. These may be classed

6 Lake & Pike: *Post-operative Tests in Labyrinthine Disease*

as latent labyrinthitis (Bárány). In Case I, which was associated with very severe attacks of vertigo, at the radical mastoid operation a distinct depression was seen over the promontory of the external semicircular canal, and in this case it is shown that complete recovery may occur, both as regards vertigo and the function of the labyrinth, as proved by the caloric or turning reactions; that as regards caloric and turning reactions more reliance should be placed on the former, as is shown in Case VI, in which ten months after the labyrinth was removed the turning reaction was equal on both sides; that in cases where the labyrinth was removed by operation, the caloric reaction, as would be expected, was negative.

With regard to nystagmus in these labyrinthine cases, in No. III. there was no spontaneous nystagmus a few weeks after the operation; in one, No. VI, there was slight right and left nystagmus. With regard to the nystagmus immediately after the operation there was usually strong nystagmus to the sound side, which gradually diminished until in a few weeks there was either no spontaneous nystagmus or slight right and left. In Case III the nystagmus was never very strong, and it disappeared in one week, whereas in Case V the nystagmus was most marked on looking towards the operated side. This is said to point to an intracranial lesion. In regard to Cases VII and VIII—so-called latent labyrinthitis—in neither of these was any spontaneous nystagmus noted. In both these cases the turning nystagmus was much below the normal on testing the affected ear.

DISCUSSION.

Mr. CRESSWELL BABER said the Section was much indebted to the authors for the series. He had recently seen Bárány do many of the tests, and he asked whether the authors had used the caloric reaction under the anæsthetic after performing the radical operation. Bárány employed it under light anæsthetics as the patient was coming to. There was on using cold water only a slow movement towards the side being tested, the quick nystagmus being abolished by the anæsthetic. Also, had the authors had any experience of the "fistula symptom" produced by condensation and rarefaction of air in the meatus in any case before operating on the labyrinth?

Dr. MILLIGAN asked what was Mr. Lake's experience of the relative value of the caloric and rotation tests.

Dr. DAN MCKENZIE, referring to the classification of labyrinthitis into "serous" and "purulent," suggested that the terms "mild" and "severe"

were preferable. He drew attention to a paper of his on the clinical value of the labyrinth tests¹ in which a modification of Bárány's caloric test was described. It consisted in a measurement of the period required to evoke nystagmus. In that paper, also, he had reported several cases of circumscribed labyrinthitis cured by the radical mastoid in some of which the vestibular sense was found to be impaired after the cure. With regard to vertigo coming on after the operation, he drew attention to that class of mild post-operative labyrinthitis where the phenomena of vestibular irritation, though well marked at the onset of the attack, rapidly disappears, leaving the labyrinth functionally intact. Alexander had recently described three of these cases. The speaker suggested that their occurrence was due to interference with protective granulation tissue on the outer wall of the labyrinth.

Mr. A. L. WHITEHEAD asked for further information. In Case II the note said "bone over external semicircular canal destroyed, and membranous canal exposed for about three-sixteenths of an inch"; did they mean that it was removed for three-sixteenths of an inch? In Case IV he said "labyrinth opened"; did he mean the complete operation, or that a portion of it was opened?

Mr. WEST took exception to the use of the terms "mild" and "severe" as an improvement on such definite statements of pathological observation as were implied in the terms "serous" or "purulent." In the latter we had at least some fixed standard by which things might be judged; in the former the standard was free to vary with the ideas of the person reporting the case. It would be difficult to say that any true case of post-operative labyrinthitis was of such a nature that serious consequences could not ensue. Broadly speaking, Mr. Lake's results seemed to agree with those which were the common property of all who had had much to do with such cases. It came to this, that where there was a simple fistula of the external canal with a functional labyrinth, the performance of the radical mastoid operation without interference with the labyrinth was in the great majority of cases adequate treatment, and these patients would, after such an operation, be left with an apparently normal labyrinth so far as tests went. But if the patient had had a general infection of the labyrinth or invasion of the vestibule before operation, or if at the time of operation the vestibule were opened, that patient would afterwards show a negative response to all tests. One met with occasional exceptions to both these generalizations; very rarely one saw cases of slow progressive infection of the labyrinth, extending from a fistula of the canal, which at the time of primary operation did not involve the membranous labyrinth, and the condition required further operation of a destructive kind for the relief of the vertigo; on the other hand, it was difficult every now and then to satisfy oneself that a patient whose vestibule had been operatively opened had really lost all power of response to tests.

¹ *Journ. of Laryngol., Lond., 1909, xxiv, p. 646.*

8 Lake & Pike: *Post-operative Tests in Labyrinthine Disease*

Mr. LAKE replied that the condition of the labyrinth in the first case was that there was a depression instead of a protuberance in the site of the external semicircular canal. He had never tested the nystagmus under an anæsthetic, and he could not see what useful purpose it could serve. In Case II the bone of the external semicircular canal was destroyed and the canal exposed. There was a fistula at each end of the canal, which was exposed after the removal of the exposed membranous canal. With regard to the relative value of the rotatory and caloric tests, he would place more reliance on the former. He had never yet had a septic labyrinth after a mastoid operation; he took such precautions as would prevent that. In Vienna he had seen the operator wash the wound out with sterilized water and pack it, even when there was a labyrinthine fistula. He (Mr. Lake) used the strongest antiseptic solution, at the time filling the cavity with Lister's or some similar strong solution. Perhaps the lack of the use of antiseptics on the Continent in such cases accounted for the frequency of labyrinthine suppuration there. The bone cavity should be treated as a septic wound. He did not think that in any of his cases there was a progressive infection. In Case IV the labyrinth was opened above, through the external canal and through the foramen ovale. Post-operative vertigo was not alone a question of infection, but he thought it must often be a form of cicatricial contraction or involvement of the vestibular and semicircular canal nerve fibres.

. Dr. PIKE, in reply, said he thought the caloric reaction was decidedly more useful than the turning. Bárány said that when the labyrinth was normal, after the turning reaction to either side, there was 25 seconds' nystagmus or more; if one labyrinth was obliterated, the other labyrinth took on part (one-third) of the action of the other side—i.e., if, say, the right labyrinth was obliterated, then after 10 turns to the right one would get about 20 seconds' nystagmus to the left, after 10 turns to the left about 10 seconds' nystagmus to the right, whereas with both labyrinths intact one would have had about 30 seconds' nystagmus to right and left. Case IV did not show 25 seconds' nystagmus, but was practically equal on the two sides. The strength of the nystagmus depended largely on how suddenly the rotation of the patient was stopped. The cases also showed the difference between fistulæ and erosion. In Vienna, where he was working in a clinique four months, he only once saw a definite "fistula symptom;" there was in the case a fistula into the external semicircular canal. It was a delicate test to do: One had to increase the pressure with an air balloon, and make the patient look steadily at the operator. If there was a strong and definite nystagmus on increasing pressure, there was a fistula into the labyrinth. If there was only slight nystagmus, it might originate in the foramen ovale or a deficiency in the foot-plate of the stapes. Was the vertigo caused entirely by the nystagmus? If one moved the eyeball with the finger from side to side, objects in the room moved in the opposite direction, and so it looked as if the nystagmus might be the cause of the vertigo. But that that was not so entirely could be proved by doing the tests on blind

people. Recently with the caloric test, in the case of a quite blind man, he got a strong caloric nystagmus. After a time he increased the amount of fluid he was using, and the patient said he felt "funny," and when made to stand up he said he felt drunk, and that either he or the room was going round. The vertigo in this case could not be said to be an ocular effect.

Dr. MILLIGAN said that probably most members preferred the caloric test, but at Copenhagen, where he recently was, the first assistant had a most ingenious rotatory chair, which could be stopped absolutely suddenly, and he noticed that at the clinique more attention was paid to the rotatory than to the caloric test.

Demonstration of Hays' Pharyngoscope.

By W. MILLIGAN, M.D.

THE pharyngoscope invented by Harold Hays, of New York, consists of a vertical and a horizontal shaft which join at right angles at the outer third. The inner portion of the horizontal shaft consists of a



Hays' pharyngoscope.

central circular tube with an electric-light carrier upon either side, the three components being incorporated in a flat piece of metal. From the inner end project two water-tight electric lamps which give an intense illumination, and which become sufficiently warm to prevent moisture condensing upon the lens, but not so overheated as to burn the surrounding mucosa. In the central tube is inserted the telescope made

upon the principle of the Otis cystoscope. Attached to the eye-piece of the instrument is a small metal ball which indicates the position of the lens at any given moment. The vertical portion of the instrument is attached to the horizontal by a screw joint, and contains the wires for connexion with a rheostat. An arrangement is provided for cutting off the current, so that the lamps need not be kept burning until the instrument is in the mouth and in position. The instrument is placed in the mouth like a tongue depressor, and in such a manner that the inner end of the telescope is $\frac{1}{16}$ in. from the posterior pharyngeal wall. When once in position the patient is requested to close his mouth and to breathe through his nasal passages. When the lens points upwards, as indicated by the position of the ball upon the eye-piece, a view of the pharyngeal vault is obtained. When rotated to the side, as indicated again by the position of the ball, the mouth of the Eustachian tube and lateral pharyngeal wall come into view. Although the instrument is useful mainly for diagnostic purposes, it can also be employed to control local applications to the mucosa of the nasopharynx when made through the nose.

DISCUSSION.

Dr. H. J. DAVIS said he had used the instrument for several months, and there were a few practical points which he had discovered. It was useful for demonstrations and for those who could not see the larynx and post-nasal space without it. It should be inserted into the mouth before turning on the light, and then the lips closed firmly. The simplest plan was to find the larynx first; then, if the telescope was rotated upwards through half a circle, the post-nasal space came into view. Sometimes the uvula, when elongated, rested on the lens, and nothing could be seen; he had noticed that if the patient's lips were then kept apart, instead of closed, the post-nasal space at once came into view. The chief objection to the instrument was the expense, and the lamps soon burnt out.

Dr. MILLIGAN replied that he could not say he had found the instrument much better than the ordinary post-nasal mirror, but it was of advantage in the examination of the larynx and post-nasal space in patients who were very ill and could not bear manipulation in the ordinary way. It was true that it was rather expensive; the instrument, as it stood, cost 6 guineas and the lamps 4s. 6d. each.

**Lantern-slide Demonstration, illustrating the Pathological
Changes found in the Ear of Deaf-mutes.**

By A. A. GRAY, M.D.

THE ears of four deaf-mutes, in all, were examined. In none of these was there a previous clinical examination, nor was the opportunity afforded of examining the brain.

Case I.—The membranous labyrinth of a deaf-mute, examined macroscopically. No deformity was present, beyond the fact that the organ was larger in size than that of the maximum hitherto recorded in the human adult normal labyrinth. In this case the outer and middle ear were quite normal. The cochlear branch of the auditory nerve was almost completely degenerated.

Case II.—The membranous labyrinth of the *left* ear of a deaf-mute, examined macroscopically. The organ was larger by 2 mm. in length than that of the maximum normal adult labyrinth hitherto examined. Calcareous salts present in the utricle and saccule. No deformity of the labyrinth visible. The middle ear was quite normal and the tensor tympani was not atrophied. The cochlear branch of the auditory nerve showed very marked degeneration. Another slide showed the hammer and anvil of the *right* side in this case. The joint was ankylosed, otherwise the middle ear was normal. The cochlea was examined microscopically, and showed in the lower and middle whorls marked depression of the membrane of Reissner and disorganization of the organ of Corti. In the upper whorl there was a curious enlargement of the stria vascularis. The cochlear branch of the auditory nerve was markedly degenerated; the facial and vestibular portions appeared healthy.

Case III.—The middle and outer ears of the *right* side were normal in every respect. The tensor tympani was not degenerated or atrophied. The labyrinth, on macroscopic examination, showed no deformity beyond the fact that it was considerably larger than the maximum normal adult labyrinth. On the *left* side the middle and outer ears were normal, and the tensor tympani was not degenerated. The labyrinth was prepared microscopically. The membrane of Reissner was depressed and thickened, but was not actually adherent to the organ

of Corti. The organ of Corti was completely disorganized throughout the whole cochlea. In the upper whorl of the latter there was the same peculiar enlargement of the stria vascularis as was found in Case II. The cochlear branch of the auditory nerve was in a marked state of degeneration, as was also the ganglion spirale. The facial and vestibular nerves were normal in appearance.

Case IV.—The right ear alone was sent for examination. The middle and outer ear were normal in every respect. The cochlea was examined microscopically, and was found to present an appearance which could hardly be considered other than normal. The organ of Corti was not disorganized, nor was the membrane of Reissner depressed. The ganglion spirale and cochlear branch of the auditory nerve appeared to be quite healthy. It is quite possible that this case was not, properly speaking, one of true deaf-mutism, but a case of mental defect.

The chief points of interest in these four cases are the normal condition of the middle and outer ear and the absence of atrophy or degeneration of the tensor tympani. The depression of the membrane of Reissner and disorganization of the organ of Corti are characteristic features of many cases of deaf-mutism, and have been recorded previously by Alexander, Denker, Görke, and others. The enlargement of the stria vascularis has also been recorded previously, as well as the degeneration of the cochlear branch of the auditory nerve.

The most interesting point, however, in the investigation of these cases relates to the size of the labyrinth. In all the three cases which were examined macroscopically the organ was not only larger than that of the average normal adult human labyrinth, but was actually larger than the maximum normal recorded. This difference in dimensions applied to all parts of the organ. In view of the fact that we would naturally expect an organ which had never properly developed physiologically to retain its small, infantile type, this fact is very surprising. I would venture to suggest that it indicates an increase in the intralabyrinthine pressure before birth, owing either to changes within the organ itself or within the cranial cavity, and transmitted from the latter by way of the aqueduct of the cochlea. In prenatal life the capsule of the labyrinth is cartilaginous for a very considerable period of gestation, and could yield to the pressure in a way that it would not so readily do in adult life.

DISCUSSION.

Mr. YEARSLEY said the cases described would have been of greatly enhanced value if Dr. Gray had received with them full clinical details, especially of the functional tests. He saw an opportunity for the Section—namely, that if Dr. Gray would draw up an account of the exact way in which he wished the specimens preserved, and if those particulars were sent to each member of the Section, possibly a larger number of cases would be sent to Dr. Gray. The difficulty of getting pathological material for deaf-mute work seemed to be very great. He (Mr. Yearsley) had recently completed a detailed functional and physical examination of 500 deaf-mutes in the County Council schools, and during that time three cases had died—two from scarlet fever and one from accident—but it was impossible to obtain their temporal bones. He had been in communication with the other doctors connected with the Council, and hoped that it might be possible to get material from cases which died. The Section might be able to help Dr. Gray's studies in the way he had suggested.

Dr. URBAN PRITCHARD said he was particularly interested in the specimen showing polypoid growth coming from the stria vascularis. It was very like the folds of tegmentum seen in a bird's cochlea; the cells surrounding it were also wonderfully like the pigment cells found in the tegmentum. Was that the result of inflammation? He thought not. Still, it was difficult to understand how that fold or outgrowth occurred. It was an interesting question whether the reduced pressure within the membranous labyrinth was the cause or the effect of the deaf-mutism, or the destruction of the internal ear. With regard to the depression of the membrane of Reissner, he would be willing to take the opinion of Dr. Gray; but one had to be very careful, because in the preparation one altered the size of the object, and there was apt to be a folding of the membrane of Reissner.

Mr. SYDNEY SCOTT expressed sympathy with Dr. Gray in his difficulties to obtain full information of the life-histories of patients whose temporal bones he had examined so carefully. He thought it was an occasion on which he might emphasize the necessity for a complete physiological and subsequent pathological examination of what he would like to term the auditory system, in such cases. This should include the results of testing both the cochlear and vestibular sense organs; they would like to know the otoscopic appearances of the membrana tympani during life, and the condition of the pharynx and Eustachian tubes. They should obtain, if possible, a record of the state of the hearing as regards "tone-range" and "tone-intensity," and the occurrence of gaps in the auditory field should be especially noted. A neurological survey should also be made to ascertain whether there are signs of any lesion which might involve projection fibres of the cochlear or vestibular nerves towards the cerebral cortex or internal nuclei. Furthermore, the conditions under which such examinations were made should be fully described, together with the state

14 Gray: *Pathological Changes in Ear of Deaf-mutes*

of intelligence and mental condition of the patient. Turning to the pathological examination, the speaker would not be content until the brain and both temporal bones had been investigated as completely as possible. It so happened that through the courtesy and with the co-operation of Dr. Robert Jones (Superintendent at the Claybury Asylum for the Insane), Mr. Scott had tested twenty-five examples of deaf-mutism, in the way indicated, only a few months ago. Within the last few weeks one of these patients had died of pneumonia; they had obtained the brain and both temporal bones within sixteen hours of death, the body having lain in the meantime in the cold chamber, temperature 0° C. Dr. Robert Jones was examining the brain with Dr. Mott, while Mr. Scott was decalcifying the temporal bones. He had found gross changes in the middle ear on both sides, but it was too early to describe these changes, as the examination of the labyrinth was not yet completed. He understood that Dr. Mott found microscopic signs of degeneration in the cortical cells of the auditory area. He hoped to furnish fuller details later. He mentioned the case on the present occasion, because he felt they should insist upon the importance of a microscopic examination of the brain as well as of the peripheral sense organs. With regard to the depression of Reissner's membrane so clearly seen in Dr. Gray's beautiful specimens, he thought they could accept this as the true position of the membrane in these cases, and that it was not an artefact as suggested by Dr. Urban Pritchard. His reasons for this view were that in sections of the normal human cochlea he had never met with such inward displacement of Reissner's or of the basilar membrane, when the ductus cochleæ was cut radially. The speaker added that all his sections had been cut in paraffin, not celloidin. Moreover, in studying the sections through the cochlea of deaf-mutes figured in the monographs of Alexander, Denker, Panse, and other continental observers, he had been struck with the frequency with which this inward displacement of Reissner's membrane had been met with. Dr. Gray had suggested a cause by supposing an increased pressure in the perilymph, secondary to increased intracranial pressure, presumably due to intraventricular distension or hydrocephalus. While admitting this possibility, Mr. Scott suggested that a diminution of pressure in the endolymph of the ductus cochleæ would also account for the inward displacement of the membranes, whether this was due to diminished secretion or increased absorption. The degeneration of the stria vascularis, judging by the proliferation of its epithelial cells, was in accord with the hypothesis of a diminished secretion of endolymph, which would favour a lower pressure in the ductus cochleæ and retraction of the two membranous walls. This view would not, however, account for the general enlargement of the labyrinth to which Dr. Gray had drawn attention.

Mr. C. G. LEE said that recently he heard a distinguished pathologist of one of the large county asylums class cases of deaf-mutism with idiots and degenerates. He felt much surprised by the statement, and was relieved when he heard Dr. Gray's remarks. His own experience was that they were

not always degenerates, but that their disease was local. The result of the alienist's view was practically to refuse education to such persons. But these children were often sharp and responded to education. From his association as medical officer with a school for deaf-mutes he could corroborate the statement as to the difficulty of obtaining pathological specimens. He felt much rewarded for having come to hear the paper.

Dr. GRAY, in reply, agreed to the similarity between the growth in the stria vascularis and the tegmentum vasculosum in the bird and reptile. He had noticed the same when he looked at the specimens. The stria vascularis was, from the point of view of evolution, the ultimate result in the mammal, of the tegmentum vasculosum of the reptile; and it was possible the human subject went through a stage in its fetal development in which the tegmentum vasculosum was present, but he did not think embryologists had yet got down to such refinements. Such a stage might be present, but be passed through with great rapidity in a mammal, and the embryologist might miss it. It might therefore be that in the specimens shown the structure was the result of arrest of development in the prenatal life at a particular time. With regard to the depression of the membrane of Reissner, he showed the normal specimen first, to indicate how it was prepared with collodium preparation; there was practically no fold. But in the others there was actual thickening and marked depression, and the same had been observed by others. A more complete examination of patients should be impressed upon physicians and surgeons in hospital wards. With regard to the reverse action of pressure, possibly the loss of function might cause a difference in pressure. It was true that deaf-mutism was a local disease, and the subjects of it were but rarely degenerates; only in recent times had aurists realized that it was not a central defect. Deaf-mutes were often very bright, and in the majority of so-called congenital cases, even, the lesion was in the labyrinth. It was but rarely a central lesion alone, though in some this might be the case.

Case of long-continued Suppuration (Aural) with Spontaneous Cure ?

By G. N. BIGGS, M.B.

THE patient came to my Out-patients' Department at the Seamen's Hospital complaining of deafness in both ears. There had been suppuration in the right ear all his life until five or six years ago, when it gradually got less and finally ceased. No headache at any time. When at school he says that he had attacks of vertigo, but cannot give any special particulars about them. Slight facial paralysis on the right side.

16 Biggs: *Long-continued Suppuration with Spontaneous Cure*

On examination, a large cavity is seen, resembling that left after a radical mastoid operation; this is completely lined by healthy epithelium. The interest of the case lies in the fact that the patient has never had any treatment for the ear, and is quite certain that there has never been any operation of any kind; this is supported by the fact that no trace of any incision can be found. Presumably, therefore, the case is one in which the disease has cured itself. Owing to shortness of time I have been unable to go thoroughly into the case (i.e., tests for hearing, &c.), but hope to bring these forward at some subsequent meeting of the Section.

Otological Section.

February 5, 1910.

Dr. EDWARD LAW, President of the Section, in the Chair.

Letter from Professor Politzer.

THE following letter was read from Hofrat Professor Politzer, in answer to an illuminated address sent by the Section to congratulate him on the fiftieth anniversary of obtaining his degree of Doctor of Medicine :—

VIENNA, *December 27, 1909.*

MR. PRESIDENT,—

I am most sincerely delighted and gratified at the cordial congratulations you kindly transmitted to me from the members of the Otological Section of the Royal Society of Medicine, in remembrance of the fiftieth anniversary of attaining my doctor's degree. I am the more sensible to the honour you confer upon me as it comes to me from a country for which I have always entertained a special feeling of sympathy and thankfulness. It was in dear Old England the immortal Toynbee guided my first steps in pathological anatomy of the ear.

I have much pleasure in stating that the wonderful work founded by Toynbee and Wilde has since developed in a most splendid way, and is daily improved and advanced by a considerable number of eminent representatives of our special science. One of them, Mr. Arthur Cheate, brought me your greetings, the signification of which was increased through this excellent and much-esteemed messenger. His coming to Vienna and reading the address before the festival assembly, and announcing my nomination as your only honorary member, will form the most beautiful remembrance for the rest of my life.

18 Scott: *Microscopical Sections through Mastoid Antrum*

Kindly give my best thanks to the members of the Otological Section of the Royal Society of Medicine in England, and believe me, Mr. President,

Your most obedient Servant,

ADAM POLITZER.

THE PRESIDENT (Dr. E. Law) said he thought the thanks of the Section were due to Mr. Cheatle for his kindness in the matter. He made a special journey to Vienna to present the address, and he (Dr. Law) proposed from the chair that the best thanks of the members be accorded to Mr. Cheatle. This was carried by acclamation.

Microscopical Sections through the Mastoid Antrum in a Fatal Case of Scarlet Fever demonstrating Streptococcus conglomeratus *in situ*.

By SYDNEY SCOTT, M.S.

THE specimens under the microscope were obtained nearly two years ago in the ordinary course of studying the microscopic appearances of infective disease of the temporal bone. The following brief note of the patient's history was obtained through the courtesy of Dr. E. W. Goodall :—

History: J. B., aged 4, was admitted to the North Eastern Fever Hospital of the Metropolitan Asylums Board on November 16, 1907, with scarlet fever. He had faucial ulceration, and subsequently developed lobular pneumonia, bilateral purulent otitis media, and acute nephritis, to which he succumbed on December 7, 1907, in the fourth week of the disease.

At the autopsy the characteristic features of the above-mentioned complications were found. There was no intracranial infection. The left temporal bone was removed and placed in formalin unopened.

TECHNIQUE.

(1) Fixative: 5 per cent. formalin at temperature about 18° C. for seven days (Zenker's fluid or Flemming's "strong" solution is, in my experience, to be preferred to formalin for fixing tissues containing

bone).¹ After-fixative: Müller's fluid without washing out formalin; temperature, 43.3° C.,—i.e., 110° F.—which happened to be the temperature of the paraffin cupboard. Two days.

(2) Decalcified in solution of phloroglucin 1 per cent., and nitric acid 10 per cent., in water. Transferred to this fluid from Müller's fluid without washing in water. Five weeks.

(3) Washed mass in running tap-water twenty-four hours.

(4) Cut mass into convenient size for imbedding.

(5) Preserved in 70 per cent. alcohol for three months—i.e., until an opportunity occurred for completing the examination.

(6) Dehydrated in 90 per cent. alcohol, several changes. Twenty-four hours.

(7) Absolute alcohol, several changes. Twenty-four hours.

(8) Cleared with xylol, several changes. Twenty-four hours.

(9) Imbedded in paraffin, melting at 43.3 C., about six hours, with four changes.

(10) Cut sections with Cambridge rocking microtome, old model, cutting in curved plane; sections 7—8 μ .

(11) Sections of the mastoid antrum were stained with methyl violet, Gram's iodine solution, and decolorized with alcohol by Gram's method. Counter-stained in aqueous safranin.

(12) Dehydrated sections in alcohol.

(13) Cleared in xylol.

(14) Mounted in Canada balsam. Date May 1, 1908.

Microphotographs of the sections and lantern slides were kindly prepared by Dr. Albert Norman, October, 1909. They show the appearance of the bone of the mastoid with the mucosa lining the antrum and accessory cells (fig. 1). The submucosa is swollen, but does not occlude the lumen of the cells. Under the microscope the tissue is seen to be in a state characteristic of intense inflammation; the blood-vessels are dilated and engorged; the extra-vascular tissues are infiltrated with leucocytes, and the superficial layers of the mucosa show no recognizable epithelium. Here and there in the mucosa and outside it are clumps of organisms which have been stained by Gram's method.

If we examine one of the masses of organisms more closely magnified, 500 diameters (fig. 2), it is possible to see chains of cocci forming a coil which has been likened to a ball of wool. This grouping of cocci into a mass can be accounted for by the extreme length of the

¹ See *Journ. of Anat. and Phys.*, July, 1909.

chains. Morphologically, the organism is evidently a streptococcus, but whether it is the specific organism of scarlet fever cannot, of course, be stated.

The well-marked conglomeration suggests its resemblance to the *Streptococcus conglomeratus*, so-called by Kurth, 1891,¹ who first drew attention to what he regarded as possibly a specific feature of streptococci isolated from scarlet-fever patients. It is, however, generally known that a streptococcus having the characters of pyogenes may assume similar conglomerate growth (vide infra). Without a knowledge of the bio-chemical properties of the micro-organism in these sections further differentiation is impossible. The organism, for instance, may be the *Streptococcus scarlatinae*, which Dr. E. Klein described in 1886-7,² or the *Streptococcus anginosus* described by Drs. Andrewes and Horder, 1906,³ found in a certain proportion of the cases of scarlet fever which they investigated; or, what is perhaps more probable, the organism may be the *Streptococcus pyogenes* as defined by Dr. M. H. Gordon.⁴ Dr. Gordon has kindly lent me some of his lantern slides of streptococci which he obtained, illustrating the bacteriology of scarlet fever. The first of Dr. Gordon's slides represents a broth culture showing the excessive conglomeration of organisms at the bottom of the culture tube. It was this feature to which Kurth drew attention. As mentioned above, others have found *Streptococcus pyogenes* clumping in this way.⁵

Dr. Gordon's sections of the tonsil⁶ and pharyngeal wall of the cervical glands, and of the spleen also, show the peculiar grouping of the cocci in the tissues. I am not aware of any sections showing the organisms *in situ* in the temporal bone removed from examples of scarlet fever. I merely add that the auditory ossicles were not necrosed, and the internal ear was intact.

¹ *Arch. a. d. Kaiserl. Gesundh. samte*, 1891, vii.

² Report of Her Majesty's Local Government Board, 1886-7. "On the Etiology of Scarlatina," with plates.

³ See "Study of the Streptococci Pathogenic for Man," *Lancet*, 1906.

⁴ "The Bacteriology of Scarlet Fever," *Practitioner*, January, 1909. (With historic review and references to Dr. M. H. Gordon's earlier researches.)

⁵ Compare illustrations in "Notes of a Case of Otitic Meningitis, with Histological Specimens (lantern slides) of the Labyrinth, demonstrating Streptococci *in situ*." (The streptococcus was not differentiated by Gordon's method in this case.)

⁶ Dr. H. M. Gordon: "Further Report (1899-1900) on the Bacteriology of Scarlatina, with Special Reference to the Investigation of Ten Fatal Examples of that Disease," contained in the Report of the Medical Officer, Local Government Board, Appendix B, No. 2. Printed by Darling & Son, London, 1901.



FIG. 1. ($\times 9$.)

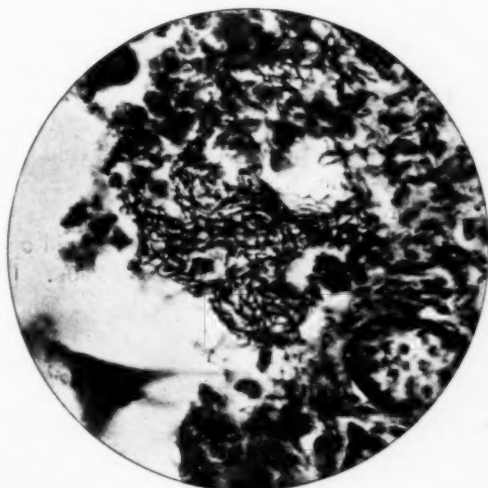


FIG. 2. ($\times 500$.)

The above subject was introduced with a hope that bacteriologists who have special facilities, and who are interested in otology and in the proceedings of this Section, would, as occasions arise, investigate the cultural features of streptococci bio-chemically by the methods elaborated by Dr. M. H. Gordon, and which I believe were first introduced to otologists by Mr. C. E. West in a short, valuable paper in the St. Bartholomew's Hospital Reports, 1907.¹ And it is important also to ascertain the appearances of the organisms *in situ* in the tissues to control the bacteriological findings *ex corpore*.

Demonstration of some Anatomical and Pathological Preparations.

By SYDNEY SCOTT, M.S.

SERIES I.

(1) Specimen of the normal membranous and periosteal labyrinth, mounted in paraffinum liquidum (Dr. Albert Gray's method). From a patient who died in St. Bartholomew's Hospital, 1908.

(2) Stereoscopic photographs of above taken by Mr. D. M. Stone.

(3) Improved glass model of the semicircular canals and utricle, adaptable for demonstration, made by Messrs. Maw and Sons' glass-blower, under supervision.

(4) Decalcified right petrous bone after healed radical mastoid operation, showing absence of stapes and replacement of the foot-plate by a translucent membrane which closed fenestra ovalis. Internal ear normal. No record of hearing available. [Reference No. 188R.]

(5) Decalcified petrous bone after radical mastoid operation from a case of lateral and cavernous sinus thrombosis, 1906. [Reference No. 191.] A coronal section of the vestibule shows the whole of the stapes displaced bodily into the vestibule. Blood-clot is seen in the vestibule and semicircular canals, but not in the cochlea.

(6) Decalcified petrous bones from a boy who died with bilateral auditory nerve tumours. [Reference No. 195.] Each bone is divided coronally and shows the growth, "glio-sarcoma," filling the internal

¹ C. E. West: "Notes on the Infection in Acute Otitis Media and Consecutive Acute Suppurative Mastoiditis," *St. Bart.'s Hosp. Reports*, 1907, xliii.

auditory meatus and invading the lowest coil of the cochlea and the vestibule. The histological sections show the growth has spread into the base of the modiolus and into the scala tympani. The ganglion cells in the zona ganglionaris have disappeared and the cochlear fibres have lost their medullary sheath. The vestibule has been invaded through the region of the saccule, which is destroyed, and the nerves to the utricle, superior and external ampullæ are in a similar stage of degeneration. The facial nerve beyond the genu retains the medullary sheath and does not present signs of degeneration. From a patient, a boy aged 15, who was under the care of Mr. Rawling, St. Bartholomew's Hospital, 1909.

SERIES II.—NORMAL ANATOMY.

Lantern-slide photograph. A portion of the adult skull decalcified *en masse*, with the brain *in situ*. The mass had been divided horizontally with a razor through the internal auditory meatus, to show the relations of the sigmoid sinus, the posterior mastoid cells, and internal ear to the cerebellum, pons, and fourth ventricle. The three usual otitic channels of infection of the cerebellum were displayed on the screen—viz., from thrombosed lateral sinus, posterior mastoid cells, and internal ear. The anatomical route for evacuating cerebellar abscess, originally advocated, he believed, by Mr. Whitehead, was likewise indicated by the exhibitor.

SERIES III.—SURGICAL IMPORTANCE OF THE REGION OF THE SACCULE.

Three histological sections of the normal petrous bone were shown on the screen by means of the epidiascope to display the relation of the saccule to the internal auditory meatus. The sections passed through the vestibule at the level of the lower, middle, and upper part of the foot-plate of the stapes. The saccule was divided below the macula, through the macula and above the macula. In each section it was seen that the inner wall of the saccule was immediately adjacent to the thin partition of bone which intervenes between the vestibule and the internal auditory meatus. The thinness of the barrier explained the ease with which the internal meatus can be opened by curetting the vestibule in this region. The perforations by the nerves of the saccule through this plate of bone would account for the spread of infections from the vestibule to the internal auditory meatus.

By histological examination of the internal ear in fatal cases of meningitis, Mr. Scott had found gross changes of an inflammatory nature in one or other labyrinth, with otitis media on the same side. He was disposed to consider that this was the usual channel of infection in most cases of otitic lepto-meningitis (*see* Series IV).

SERIES IV.—THE RESULT OF HISTOLOGICAL EXAMINATION OF THE
PETROUS BONES IN MENINGITIS.

*Microscopic Section (epidiascope) and Microphotographs (lantern slides)
of the Infected and Non-infected Labyrinth, selected from Twenty-
eight Cases of Meningitis.*

The investigation formed part of a general inquiry into the post-mortem appearances of the internal ear, derived from an examination of some 200 pairs of petrous bones. Death was due to various causes. Uncomplicated meningitis was present in twenty-eight instances (this excludes cases of meningitis associated with brain abscess, lateral sinus thrombosis, diseases of the nose or orbit and fractures of the skull). In these twenty-eight cases the infection was typical miliary tubercle in eight. The internal ears were decalcified and found to be free from disease. In one case (No. 148) there was tuberculosis of the middle ears. Caseous masses, spreading through the tegmina tympani, involved the dura mater of the middle fossæ; the masses produced indentation of the temporo-sphenoidal lobes, but no adhesions were present, and the brain did not contain any tuberculous deposits. Typical miliary tubercle was present in the pia-arachnoid of the base, especially along the middle cerebral arteries. The temporal bones were removed from a child who died with general tuberculosis in the Evelina Hospital for Sick Children in 1908. Attempts to stain the tubercle bacillus in the sections were unsuccessful. Nevertheless, nothing could be more typical of the naked-eye appearance of tuberculosis in the various organs of the body of this child. Among the non-tuberculous cases of cerebrospinal meningitis, in two the middle ears were normal (specimens shown). In the remaining sixteen cases the middle ear was in a state of suppuration, acute or subacute, and unilateral or bilateral; in eleven of these cases (specimens shown) the internal ear, on one or other side, was also the seat of diffuse infection. The internal ear lay in the channel along which this infection could pass, from the middle ear to the pia-arachnoid

perineural sheath of the eighth nerve in the internal auditory meatus. Thus, of meningitis, twenty-eight cases:—

(A) *Tuberculous*, eight cases: Diffuse lepto-meningitis secondary to remote source of infection.

(B) *Non-tuberculous*, twenty cases: (1) Diffuse cerebrospinal meningitis, eighteen cases; (2) Local otitic meningitis, two cases: No. 186, posterior fossa, from obvious mastoid infection; No. 188, middle fossa, from visible infection through tegmen tympani.

CONDITION OF MIDDLE EAR IN EIGHTEEN CASES OF CEREBROSPINAL MENINGITIS.

				Cases No.
Seven acute or subacute otitis media (unilateral)	{	75, 90, 163, 164, 176, 180, ¹ 184
Six chronic otitis media	Four unilateral	73, 154, 197, 182
	Two bilateral	52, 138
Two otitis media absent	147, ¹ 149 ¹
Three doubtful (partial examination)	25, 137, 196

Excluding from consideration the last five *non-otitic* and doubtful cases, we have thirteen *possible* cases of *otitic* meningitis. There were reasons for regarding the meningeal inflammation in Case 180 as non-otitic: the middle ear being affected late in the course of the disease, the internal ears were both free from the usual signs of inflammation; a pathogenic organism, a small Gram-negative bacillus (not tubercle) was, according to Mr. Gow, isolated during life from the cerebrospinal fluid on two occasions. The child lived nineteen days after the onset of meningitis. Of the other twelve cases, infection of the labyrinth on the same side as the middle-ear disease occurred in all except one, in which case the pia-arachnoid was known to be infected as the result of an exploration of the cerebellar fossa.

SUMMARY.

It would therefore seem that the source of the infection of the meninges could be accounted for in almost every case. When the infection passed through the tegmen, the meningitis was localized in the temporo-sphenoidal fossa. When the infection proceeded directly from the mastoid cells to the cerebellar fossa it was again localized, and not

¹ A peculiar fibrinous exudate was found, with only a few cells, in the endosteal layers of both internal ears, especially near the bases of the modiolus, in these three cases (147, 149, and 180). It seemed as if this almost non-cellular deposit was the result of the neighbouring inflammation in the meninges of the seventh and eighth nerves, for the condition was bilateral.

diffuse or bilateral. In the cases in which the channel of infection appears to have been represented by the internal ear (eleven cases) meningitis was typical post-basal, pia-arachnoid, or lepto-meningitis. The material exhibited was obtained from Cases No. 25,¹ 52,² 90, 147, 148, 149, 163, 164, 176, 177, 180, 182, and 184. The sections from Case 163 showed organisms (streptococci) in the disintegrated membrana secundaria.

In conclusion, the speaker submitted that the material which he had collected formed part of the evidence that diffuse basal lepto-meningitis complicating middle-ear disease was preceded by labyrinthitis, and, although this view differed from the acknowledged teaching on the subject, he was disposed to think that the internal ear, and not the tegmen, was the more usual route of infection in diffuse otitic lepto-meningitis.

N.B.—As before mentioned, only cases of "uncomplicated" meningitis are under consideration.

DISCUSSION.

Mr. A. L. WHITEHEAD said the demonstration had been enjoyed immensely, especially the one slide which demonstrated so clearly the relation of cerebellar abscess to certain portions of the middle ear and its adnexa. That specimen would show, if any demonstration were needed, the absolute importance of always approaching a cerebellar abscess through the area which had so frequently been spoken of as the desirable route—i.e., the area of bone which was the inner portion of the posterior wall of the mastoid, between the posterior semicircular canal and the lateral sinus. The members could not see too many such specimens, which impressed upon one the relations of the parts upon which the otologist had to work.

Dr. A. BRONNER asked, in regard to the case of scarlet fever, what was the condition of the ear. Was there mastoid trouble or otorrhœa?

Mr. C. H. FAGGE asked Mr. Scott whether his observations on the middle ears of these cases of meningitis in children coincided with the observations made years ago by Dr. Still at Great Ormond Street, that within the ears of normal infants there was found at the post-mortems exudation of a purulent nature; whether he had found such exudate within normal ears, and, if so, whether it

¹ No. 25 has a child aged 20 weeks "brought in dead" and found to have chronic internal hydrocephalus.

² No. 52 was a case of meningitis serosa maligna.

contained organisms. He (Mr. Fagge) was not at all convinced of the relationship between those ear cases and the meningitis of which Mr. Scott had spoken, or that the path by which it was suggested meningitic infection took place from the middle and internal ear was the true one. The mere finding of infective foci in the middle ear and the meninges was no proof of the sequence of events, though it was suggestive.

Mr. A. CHEATLE said that children not infrequently died with meningitis with an intact membrane and the middle ear containing muco-pus. Some years ago, at the Society, Mr. Ballance spoke of Dr. Still's researches, and said it was normal that a child should die with purulent pathogenic material in the middle ear.¹ He (Mr. Cheatile) objected at the time, saying that he did not think it was normal for a child to die with such material in its middle ears, and all his examinations had proved to the contrary. He had examined many middle ears in children who had died, and they did not contain muco-pus. His idea was that in those patients who died with septic meningitis of the middle fossa with pus in the middle ear, with or without an intact membrane, the pathway of infection was through the peri-lymphatic spaces, connecting the middle ear with the meninges via the petro-squamosal sinus.

Mr. SCOTT, in reply, thanked the President and members for their reception of his contribution. With regard to Dr. Bronner's inquiry with reference to the scarlet-fever case, there were no changes in the ossicles of the middle ear—i.e., no caries and no necrosis. The mucous membrane of the middle ear was very much swollen and engorged, and the tympanic membrane was perforated. Mr. Fagge's question was directed to one of the knotty questions in aural pathology—viz., the significance of the presence of muco-pus in the tympanum of infants who died from various disorders. It was a matter which had puzzled Continental as well as British and American observers, and he was not prepared at present to state any new points on the subject. He appreciated Mr. Cheatile's views with regard to otitic meningitis, and he unhesitatingly accepted these views until he found evidence of unsuspected infection of the internal ear on one side in a patient who died of diffuse meningitis. This had led him to make further search, with the results he presented for consideration. He asked Mr. Cheatile whether in his cases he found any disease of the internal ear, or whether he could exclude internal-ear infection in those instances in which death had resulted from middle-ear disease with basal lepto-meningitis.

Mr. CHEATLE, replying to Mr. Scott, said he did not examine the labyrinths microscopically, but macroscopically they were healthy. In cases in which the middle ear had been full of that muco-pus he had had sections made of the lining membrane, and found evidence of disease, showing it was not a normal condition.

¹ *Trans. Otol. Soc.*, 1900, i, p. 68.

**Deafness and Discomfort in the Right Ear as Early Symptoms
in a Case of Epithelioma originating near the Right
Eustachian Tube.**

By EDWARD LAW, M.D.

THE patient, a man aged 37, was first seen on February 18, 1909. He noticed deafness in the right ear nine months previously, with the feeling of something like a "spirit-level" in the ear on bending the head to the right side. Three months' treatment as an out-patient removed the feeling of something moving in the ear, but the deafness gradually increased with occasional earache, and when walking he "could feel himself walking up his right side."

Examination: Right ear, impacted cerumen; general catarrhal condition associated with deflected septum and spurs in both nostrils. Dyspepsia. Usual tests pointed to middle-ear deafness. A catheter could not be passed, and no air appeared to enter right ear on Politzerization. March 8: Severe pain in the right ear. Patient not seen again until May 13; in the meantime, the spur in the right nostril had been removed. He now complained of frequently recurring neuralgic pain in the ear and right side of the face, occasional bleeding from the right nostril, and peculiar sensations in the right side of tongue and lip. An enlarged gland could be felt on the right side of the neck; the palate was drawn to the right side; posterior rhinoscopy revealed an indistinct swelling in the neighbourhood of the right Eustachian tube, and, on digital examination, a soft growth could be felt, which bled when touched. Mr. Tilley examined the patient and confirmed the serious nature of the disease. Potassium iodide and hydrarg. were prescribed, and admission into hospital advised.

The patient was admitted into University College Hospital on June 3, and Mr. Wilfred Trotter has very kindly permitted me to have the following notes. Operation performed by Mr. Wilfred Trotter on June 4: After a preliminary laryngotomy, the usual incision for removal of the upper jaw was made; the malar process, ascending process of the superior maxilla and hard palate divided, and the upper jaw turned outwards, so that a full and very satisfactory view of the nasopharynx was obtained. The tumour was seen to be growing in the wall of the post-nasal space between the lateral recess and the Eustachian tube, the

opening of which was invaded. The growth, together with a surrounding border of healthy muscular tissue and cartilage of the Eustachian tube, was removed, the upper jaw turned back into position and secured with a short silver wire passed through the maxillary bones just above the incisor roots. On July 2 the infected cervical glands were removed from the neck.

The patient still suffers from severe neuralgic pains in the ear and right side of the head. The deafness is stationary, but the handle of the hammer has gradually changed its position until it passes almost directly backwards.

DISCUSSION.

Dr. LAW added that when he put the case down he thought the affection uncommon, but had since learned that a number of similar cases had been recorded in the last few months.

Dr. PATERSON asked what the experience of members had been with regard to the after-history of such cases. In this case the severe pain was still present. In two cases he had seen, and which were not surgically treated, pain was the most prominent symptom. The surgeons who saw the cases had the impression that the growth could not be successfully removed, and that in the cases which had been so treated the relief was only temporary.

Mr. HERBERT TILLEY said he had seen four such cases of primary disease in the nasopharynx during the last seven months. In three there was a secondary infection of the glands of the neck. The first case of that series was the present one, and the symptoms in the other three were very similar. There was some earache and neuralgic pain on that side of the head, which distressed the patient more than his deafness. In one of the cases, which he saw in consultation, the deafness was associated with nasal obstruction and a discharge of glairy mucus from the nose. There was an ulcerating mass in the lateral wall of the nasopharynx. In the third case there was deafness and a discharge from the nose. The discharge came from a growth in the right lateral wall of the nasopharynx. Mr. Trotter had operated in three of the cases, and about four weeks later removed the gland in the neck. None of the patients, except Dr. Law's, had suffered from neuralgia after the operation. His own experience seemed to show that primary malignant disease in the nasopharynx was more common than was usually thought. Some of them sought advice on account of enlarged glands in the neck. The present case taught the important lesson that in all patients suffering from slight deafness there should be a careful examination of the nasopharynx. In the four cases the speaker referred to there was found to be paresis of the soft palate on the side of the lesion. In Dr. Law's patient the right ear was involved and the palate drawn

up to the left side owing to the paresis of the right palatal muscles; there was also some numbness about the lip, showing that the growth had affected the nerve supply to those parts. When the finger was passed into the nasopharynx it often gave more information than did the mirror. Ten days ago there was no recurrence apparent in the present case, and even the severe neuralgia was not beyond treatment; he believed Mr. Trotter proposed to inject alcohol into Meckel's ganglion, and, if that did not relieve the symptom, he would perhaps remove the Gasserian ganglion. The great point was that the life of the patient was saved. Referring to the operation, he remarked that when the jaw was swung out it was extraordinary what a good view was obtained of the posterior regions of the nose and nasopharynx. If such cases could be dealt with before extensive metastatic deposit occurred in the glands of the neck they were very successful, and the look-out was a hopeful one.

Dr. H. J. DAVIS reminded members of the Section of the case which he showed¹ nine months ago of a man with a large epithelioma of the middle ear, which had been operated upon six times, leaving an enormous cavity, at the bottom of which one could see the internal carotid pulsating. He also at times complained of intense neuralgia, and it was always relieved by curetting the part and application of the X-rays through a tube. The patient eventually became insane and died. He was 35 years of age.

Mr. C. H. FAGGE said that, looking at the matter from the surgical aspect, he was sure it would be agreed that the common cause of enlarged cervical glands in a man aged over 40 was epithelioma. It had been his practice during the last five years in every case of the kind presenting itself as a general surgical out-patient, or in the surgical wards, to examine the nasopharynx either with the post-nasal mirror or digitally. And in that routine examination he had found only two cases of primary epithelioma of the nasopharynx among a considerable number of cases of epitheliomatous glands in the neck, proved microscopically, for which a primary cause was either found or suspected to exist in the lower part of the pharynx or in the sinus pyriformis. In his experience it was much rarer to find the primary source in the nasopharynx than he had anticipated. One case was the relative of a colleague; he was brought because he was deaf and had a mass in the neck. There was a primary epitheliomatous ulcer in the nasopharynx, involving the Eustachian tube, a large mass of glands beneath the sterno-mastoid, and adherent to the deep structures, and obstructive Eustachian deafness. The pain was only trivial. The patient saw other surgeons on the question of the operation, which he (Mr. Fagge) suggested—viz., osteoplastic resection of the upper jaw. But the balance of opinion was against operation, as the disease was so advanced, and the operation was not done. The patient lived in comparative comfort three or four months, and then died of pneumonia. The other case Mr. Fagge had lost sight of. He suggested the neuralgic pain in Mr. Trotter's case, which

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Otol. Sect.), p. 89.

was not an uncommon sequel of osteoplastic resection of the upper jaw, was due to callus, and could be overcome by resection of the second and third divisions of the fifth nerve, or of the Gasserian ganglion and the divisions of the nerve arising from it, after the method advocated by Mr. Hutchinson.

Mr. HERBERT TILLEY explained that he did not mean to imply that such cases were common, but that careful examination of the nasopharynx showed them to be more common than had been thought. The neuralgia in this case was intense before the operation, and so he did not think it was due to post-operative callus.

Mr. WHITEHEAD said that in his experience sarcoma was more common than carcinoma in being a primary cause of ear symptoms.

Dr. DAN MCKENZIE said that about two years ago, at the Central London Throat and Ear Hospital, two cases came up within a few days of each other. The men complained of discharge from one ear, which was painless and had been in existence only a short time. In both there was endothelioma of the nasopharyngeal space. One of the cases was under the care of Mr. Stuart-Low. The speaker had seen them both, and they emphasized the great importance of examining the nasopharyngeal space by the mirror or the finger in all cases. The point of these cases was that the discharge from the ear was without pain.

Mr. WESTMACOTT asked whether there were many glands found in the case. In epithelioma of the Eustachian tube one would not expect to find so many cervical glands enlarged as in cases arising from secondary infection from malignant disease lower down. And from analogy with suppurative conditions around the Eustachian tube one would expect the gland below the ear to be the first affected, and more so than the other glands in that region.

Dr. BRONNER recommended in difficult cases Hays' pharyngoscope, which could be used easily and without discomfort. The nasopharynx should be carefully examined in every case of ear and throat disease.

The PRESIDENT, in reply, said that on March 8 there was nothing in the nasopharynx, and it was quite easy to examine the patient, who suffered from bad septal deflection and spurs. He was sent into hospital to have them removed, and was not seen again for two months, by which time the whole condition had changed. The patient now complained of peculiar sensations in his tongue and at the side of the jaw. There was paresis of the palate, and the mirror showed a swelling in the neighbourhood of the Eustachian tube. An enlarged gland had also appeared in the neck. The pain must be regarded as a serious symptom, but a favourable point was that, in spite of the severe pain, since the operation the patient's health had been gradually improving. With a recurrence, one would have rather expected the reverse to be the case. In answer to Dr. Paterson, he had had no experience of the future history of such cases, but he should be pleased to report further to the Section, and no doubt Mr. Tilley would do the same.

Case of Endothelioma of the Temporal Bone.

By J. DUNDAS GRANT, M.D.

THE patient, a boy aged 6, was first seen by me in November of last year on account of a swelling over the right mastoid, in which there were three discharging sinuses. There was also a hard gland to be felt below the mastoid process, and extreme narrowing of the external auditory meatus, which was full of pus; the soft parts over the mastoid were boggy and œdematous, but presented a peculiar density on palpation and a fiery redness on inspection, which, combined with a history of long evolution and of several previous operations, led me to form a provisional diagnosis of sarcoma. He had been operated on in 1907 on account of a swelling below the ear; he was then detained in hospital for about four months; he had subsequently had three operations of a scraping character, and it was reported that there was no discharge from the ear before the operation, but it had been profuse ever since. There was no history of tuberculosis.

He was admitted a fortnight after I first saw him. I made a semi-lunar incision behind the sinus-bearing skin, which I excised. I then came upon an oval cyst-like, translucent swelling, occupying what appeared to be a backward extension of the bony external auditory meatus. When this was removed, the meatus was found large enough to receive the tip of the thumb, and the floor was almost non-existent. The enlarged gland was dissected out, the antrum opened, and the greater part of the mastoid removed, the lateral sinus being exposed for about $\frac{1}{2}$ in. with pus in the groove; the wall of the lateral sinus was extremely white. An incision was made in the membranous meatus, and a drainage-tube was inserted in the mastoid opening. The microscopical examination showed the tumour to consist of typical endotheliomatous tissue. The wound, as might have been expected, did not heal cleanly, there being some inversion of the anterior lip of the mastoid incision, while flabby granulations formed freely on the posterior one. A discharging sinus remained at the lower part, in which, no doubt, sloughing was taking place; under the action of boracic fomentations this cleared up to some extent. On January 14 I reopened the wound for the purpose of bringing the lips of the upper part into better position. I scraped away the granulations and formed a large

tongue-shaped flap from the posterior wall of the meatus. The incision was closed with catgut stitches after a certain amount of undermining of the edges of the wound so as to make them freer. Union has taken place to some extent, but not completely.

DISCUSSION.

Dr. P. McBRIDE asked whether the nasopharynx had been examined in the case. He remembered an interesting case, which, however, was incomplete. A patient came complaining of his ear. He had a polypoid growth, and on examining the nasopharynx he found a growth there, which turned out to be endothelioma; and, as far as he remembered, pieces removed from the meatal growth were also endotheliomatous. The case came to an abrupt end, because the patient left the ward surreptitiously, being a Reserve man, to go to the war. But he was stopped in London, and he believed that at the Military Hospital he had had a mastoid operation.

Mr. A. CHEATLE asked whether Dr. Grant knew where the growth started. He (Mr. Cheadle) recorded the first case of endothelioma of the auditory apparatus some years ago. The patient was a lady from whom he removed the whole meatus and concha. She went on very well until two years ago, when there was found to be fullness where the tragus had been, and there was a slight recurrence. That was removed, and she was still going on satisfactorily. The tumour was evidently of very slow growth, and had started in the cartilaginous meatus.

Dr. PATERSON asked about the history of previous discharge in such cases. He hoped to report to the next meeting a case in which there was a very long history of discharge. The patient was aged 26, and the history pointed to acute symptoms only four months before her death.

Mr. FAGGE said it was not clear to him after seeing the section that it was an endothelioma, and suggested that it might be referred to the Pathological Committee.

Dr. GRANT replied that there was no evidence of disease in the nasopharynx when the boy was admitted, but he had not explored it since the operation. The disease seemed to have started in the posterior wall of the meatus, which was eroded, perhaps as much by pressure as by extension of the disease in the bone itself. The growth was of about the size of a date-stone, and shiny; white in colour. He would be glad to show it another time. There was a long history of suppuration. Dr. Wyatt Wingrave had made the pathological report upon it.

Some Features of the Auditory Apparatus of a 16 mm. Human Embryo, as shown in a Reconstruction Model (by the Wax-plate Method of Born).

By G. J. JENKINS, F.R.C.S.

EXTERNAL EAR: The embryonic tubercles of the pinna have fused, but are still indicated. The external auditory meatus is expanded internally. In the model the plug of epithelial cells has been removed to show the form of the meatus.

The labyrinth: The vestibule is compressed laterally and is somewhat quadrilateral in form. The semicircular canals are in an advanced stage of development compared with the cochlea, which is represented by a simple tube turning on itself at its tip. The cochlear element of the labyrinth lies close to the roof of the pharynx, considerably internal to the orifice of the primitive Eustachian tube, and comparatively close to the sagittal plane. The ductus endolymphaticus is large and long.

The region of the middle-ear tract: The Eustachian tube, tympanic cavity, and antrum are represented by the first pharyngeal pouch in its more or less primitive condition. Superiorly, the pouch is forming an acute fissure, and at its outer extremity is turning upwards to invade the mesoblast between the labyrinth and the external auditory meatus. Incus, malleus, and Meckel's cartilage are represented as one continuous mass. Microscopically Meckel's bar is cartilaginous in the lower part, but malleus and incus are still in the mesoblastic state and differentiated only by concentration of cells. The process from the bar in backward direction is the great process of the malleus. The incus has a thin process backwards and inwards, to fuse with the mesoblastic anlage of the stapes. The articulations have not yet been differentiated. The hyoid bar—in a prechondral stage—is seen passing upwards on the inner side of the facial nerve. The upper extremity of the hyoid bar bifurcates, the anterior portion being directly continuous with the stapes. The facial nerve lies in the fork so formed. There is a large vein lying to the outer side of the horizontal portion of the seventh cranial nerve, to the inner side of the malleus and incus, and below the external semicircular canal. It is connected posteriorly with the internal jugular vein, and anteriorly with a venous plexus internal to the Gasserian

ganglion. There is another large sinus channel, in the position somewhat of the petro-squamosal sinus, lying above the semicircular canals.

Mr. Jenkins also showed sections (10μ) of a chip removed at a complete post-aural operation from the mastoid below and to the outside of the antrum, showing columnar cells of the lining membrane of the cells of the suture between the squamo-zygomatic and petro-mastoid elements of the mastoid process. He also showed specimens of sections (10μ) of the normal adult mastoid, in which pavement epithelium lined the air-cells.

DISCUSSION.

Mr. SCOTT said he had the privilege of being acquainted with Mr. Jenkins's splendid work, which represented a move in the right direction; and he hoped that ultimately they would be able to reproduce specimens of pathological conditions by the same process.

Mr. JENKINS, in reply, said the magnification in this case was 50 diameters. The ossicles at that stage were simply concentrated mesoblast. The stapes was ring-like, the incus was particularly small, and the malleus large. The model was of a period before the synovial cavities in the body appeared. There was a large vein passing across the surface of the labyrinth in the position of the middle-ear tract, and he had not found such recorded in any book on embryology. Probably the anterior part of that vein persisted through life, but it was seen comparatively late in foetal life.

Pulsating Angioma (*Angioma racemosa*, Cirroid aneurysm) of the Right Auricle.

By GEORGE WILKINSON, F.R.C.S.

THE patient was a woman, aged 38. Her right ear had always been larger than the left, and had increased rapidly in size during the last two years. She had had severe hæmorrhage from the ear a few days before coming to the Sheffield Royal Hospital on April 27. The ear measured $3\frac{3}{4}$ in. from above downwards, $2\frac{3}{4}$ in. from side to side, and projected $1\frac{1}{4}$ in. from the side of the face. It was of a mottled red and purple colour, with many dilated vessels in the skin, and throbbed with a noticeable expansile pulsation with each heart-beat. Many large arteries could be felt entering the mass and coursing in tortuous curves under the skin.

Operation, May 3: Preliminary ligature of the external carotid. An incision carried all round the ear, and all the vessels entering from the outside ligatured and divided. The skin dissected up from the back of the auricle, and the main mass of the angioma dissected out. Very little bleeding.

A plastic operation was done on September 7 to reduce the size of the auricle. The skin was incised along the margin of the auricle and dissected up from the cartilage, which was trimmed away. Much of the connective tissue of the lobule was dissected out. The skin-flaps were trimmed and folded over in the upper part of the ear to form a new helix, and sutured.

Mr. WILKINSON reminded the Section that a similar case had been reported by Mr. Hugh Jones at a meeting of the Otological Society on March 5, 1906. A photograph of this case appeared in the *Journal of Laryngology* for April, 1906.¹ Mr. Jones amputated the ear. Mr. Secker Walker mentioned a similar case of his own in the discussion on Mr. Jones's case. The swelling in this case disappeared after ligature of the external carotid. Dr. Francis Stewart, of Philadelphia, reported a similar but somewhat more extensive case in the *Annals of Surgery* for October, 1902.² Dr. Stewart performed an operation similar to the one employed in the case here reported, but omitting the preliminary ligation of the external carotid. The operation was attended by serious loss of blood. In Mr. Wilkinson's case the external carotid was obviously enlarged. Its ligature, as the first step of the operation, prevented any considerable bleeding during the dissection of the tumour.

Objective Clicking Sound in Left Ear.

By HERBERT TILLEY, F.R.C.S.

MRS. G., aged 30, sought hospital advice for a "clicking noise in her left ear, which her doctor could also hear." She had noticed it for twenty months, and it followed an attack of influenza.

Examination (December 9, 1909): The clicking sound was regular, 120 per minute, and could be heard 2 ft. to 3 ft. from the patient's left ear. It was less easily heard when the examiner's ear was placed near to the open mouth of the patient. A rhythmic contraction of the muscles forming the floor of the mouth and of the soft palate was also noticed, and these were synchronous with the clicking sound. The noise and the muscular contractions cease when the patient holds her breath. Hearing

¹ *Journ. Laryngol., Rhinol. and Otol.*, 1906, xxi, p. 192.

² *Ann. Surg., Philad.*, 1902, xxxvi, p. 453.

is normal. No evidence of organic disease of the central nervous system. At times the clicking noise ceases altogether, but the muscular contractions continue.

DISCUSSION.

The PRESIDENT said he never remembered having seen such an aggravated and peculiar case.

Dr. PATERSON said he had a similar case about three years ago, in a married woman, who had suffered for nine months from twitching sounds in both ears. She said they "made her jaws tired." Her hearing was excellent, the clicking was rhythmical, quite audible on both sides to the bystander, and not synchronous with the pulse. On the right side it was but slight, and visible movement of the drum doubtful. In the left ear, on the other hand, there was a definite visible twitching movement of the lower and posterior part of the membrana tympani near the insertion of the handle of the malleus, and that was coincident with the clicking sound. The noise always ceased and the visible movement of the drum stopped when the mouth was open: therefore it was impossible to say whether there was a rhythmic twitching of the soft palate. On passing Valentin's salpingoscope through the nose into the nasopharynx, he thought he could make out some slight rhythmic contraction. But the mere opening of the mouth seemed to put the musculature on the stretch, and stop it. The after-history of this patient was that it persisted for some months and was replaced by twitching in the left eye and sometimes in the left arm and leg. It occasionally recurred for a short time, but always yielded to a course of iron tonics, and the patient is now well.

Dr. DUNDAS GRANT said the case was a very exaggerated one of clonic spasms. The few which he had seen before had simply a movement of the palate, not of the tongue also. It would be agreed that there were contractions of the tensor palati and tensor tympani. That clicking sound in the Eustachian tube some people could produce at will, and that was characteristic of those functional, almost hysterical, motor spasms. In this case it seemed to be due to a general disturbance of the nervous system brought about by influenza. Perhaps it originated as a habit spasm, and, in any case, it was difficult to get rid of.

Dr. McBRIDE said the present case was in some respects unique. He had seen neurasthenic patients with obvious twitching of the palate associated with clicking sound, but what he had not seen before, and what he did not think had been previously described, was the twitching of the floor of the mouth. He thought the click was probably caused by the sudden drawing asunder of the lips of the Eustachian tube. He was interested in the condition because at will he could produce a sound by contracting the tensor palati, and possibly the tensor tympani, but others had been unable to observe movement of the

tympanic membrane during the process. It was difficult to be sure of movements of the membrane, as a little displacement of the speculum was liable to simulate a movement of the membrane. The only satisfactory test was the manometer, and in the cases where that test was applied there seemed to be no movement.

Mr. HERBERT TILLEY, in reply, said that such cases belonged to the group of "clicking tic," and Schlesinger was the only neurologist who had discussed that class of case from the point of view of prognosis. He thinks that such cases almost invariably end in a lunatic asylum, showing that there was some graver central condition than might be generally supposed. Dr. Henry Head had also given a very grave prognosis in this patient. The speaker cast his memory back to a somewhat similar case (clonic contraction of pharyngeal muscles) he had previously seen, which occurred in a man whom he showed before the Laryngological Society.¹ That patient died of general paralysis of the insane four years afterwards.

Case of Tabetic Deafness.

By DAN MCKENZIE, M.D.

THE patient, a male aged 42, came to hospital a few weeks ago on account of deafness. It was observed that he was suffering from hoarseness, which, on examination, proved to be due to complete paralysis of the left vocal cord. The left side of the soft palate is also parietic, and it is possible to see a pulling-over of the left half of the posterior pharyngeal wall when he is asked to phonate, &c. Within the last few days he has begun to experience some difficulty in swallowing. The condition of the ears is a little complicated. Four years ago a radical mastoid operation was performed upon the right ear, and there is now a large cavity, on the walls of which some granulations are still present; but both the hearing and the vestibular sense are more active in this ear than in the left, which has never been affected with purulent disease. The hearing tests are as follows:—

	Right				Left
Watch	= contact	— ∞
Whisper	= — ∞	— ∞
Conversation	= 4 in.	— ∞
T. F. (256).					
Meatus	= — 30 seconds	— 50 seconds.
Mastoid	= — 10 seconds	— 20 seconds.
Rinné	= —	—

¹ *Proc. Laryng. Soc. Lond.*, 1900-1, viii, p. 51.

Weber lateralized to the right. Galton's whistle not heard in either ear. Vestibular reactions: There is no spontaneous nystagmus, or, at the most, only a very minute twitch, on extreme deviation. Caloric (cold, 22° C. to 24° C.): Right, nystagmus marked in 20 seconds; vertigo; Left, very slight nystagmus in 50 seconds, no vertigo. That is to say, the vestibular reaction on the right (the operated) side is fully equal to normal; while, on the left, it is decidedly subnormal. The pupils are unequal; the left is a little dilated and fixed, and the right, smaller than the left, shows a sluggish response to light. Knee-jerks normal; ankle-jerks absent. No ataxy.

Dr. Purves Stewart, who has examined the patient, is of opinion that the case is probably one of tabes, with involvement of the cranial nerves. The particular interest of the case to the otologist lies in the asymmetrical character of the deafness and impairment of the vestibular system. The patient is under treatment by pot. iodid.

DISCUSSION.

Mr. WILKINSON asked whether Dr. McKenzie found the method of applying the caloric test altogether satisfactory in cases in which the ears of the two sides were not in the same anatomical position. In six or eight cases in which he applied that method after radical mastoid operation, or in which the membrana tympani was absent through suppuration, the induction period of the nystagmus seemed to be shortened on the side on which the tympanic wall was exposed. In such cases he thought the duration period of the nystagmus would give a better comparison between the two sides than the induction period.

Dr. DAN MCKENZIE, in reply, said that the disks were normal in this case. It was true that exposure of the labyrinth to the direct impact on the external wall must evoke a more rapid response to the testing fluid, but where the labyrinth was involved in disease, or where it was impaired, the fact that the labyrinth was exposed would not shorten the induction period materially. If, on applying the caloric test to any case such as that mentioned by Mr. Wilkinson there was a normal reaction short of the normal period, it might be taken to mean that there was no interference with the vestibular sense on that side. He thought that the measurement of the duration of nystagmus after the caloric test was quite unreliable.

An Acoustic Aid for Persons partially Deaf from various Causes.

By H. MACNAUGHTON-JONES, M.D.

THIS was a simple appliance, and readily adjustable. A thin, sliding metal spring band connected two hollow and light imitation tortoise-shell auricles, fitting behind the pinna. The light connecting band is



divided into two halves held together by a tiny screw, so that it is easily adapted to a head of any size. For those who suffered from the degree of deafness he mentioned, it would be found most satisfactory. He was indebted to Mr. Hawksley for making the appliance for him, to which he had given the name of "Autophone" for want of a better. The principle, as he had already said, was nothing new; it was as old as the prehistoric habit of primitive man of placing his hand behind his ears to direct and collect sound-waves. A modification can be had which hooks on to the ear so as to avoid the necessity of taking the hat or bonnet off in visiting or in a public building.

Otological Section.

March 5, 1910.

Dr. E. LAW, President of the Section, in the Chair.

Twenty Specimens of Chronic Middle-Ear Suppuration and its Sequelæ, Eighteen of the Bones being of the Infantile types and Two Cellular.

By ARTHUR H. CHEATLE, F.R.C.S.

INFANTILE TYPES.

- Nos. 1 and 2.—Healed.
- Nos. 3, 5, 6, 7, 8, 9.—Discharging at death; death from other causes.
- No. 10.—Labyrinthine involvement. Death from meningitis. Operation.
- No. 11.—Death from meningitis, per posterior fossa.
- No. 12.—Death from meningitis, per posterior fossa.
- Nos. 13 and 14.—Death from temporo-sphenoidal abscess.
- No. 15.—Death from temporo-sphenoidal abscess and meningitis.
- No. 16.—Death from cerebellar abscess and thrombosis of the lateral sinus.
- No. 17.—Death from lateral sinus thrombosis.
- No. 18.—Death from other causes several years after operation for extradural abscess in posterior fossa.

CELLULAR TYPES.

- No. 1.—Healed.
- No. 2.—Caries in middle and posterior fossa; the precise cause of death is unknown.

At a meeting of the Otological Society of the United Kingdom, held on May 4, 1907, I showed ninety-six specimens of what I called the

"infantile" types of the temporal bone, and I then stated how very frequently they were met with when operating for chronic suppuration and its sequelæ, and suggested that the anatomical conditions found therein were responsible for the persistence of suppuration from the middle-ear tract. Since then I have particularly noted the type of bone when operating for chronic suppuration and its sequelæ, and found, almost invariably, the dense outer antral wall and absence of mastoid cells characteristic of the infantile types. Mr. Jenkins has made microscopical sections of pieces of the dense outer antral wall removed at operation, and was unable to discover any evidence of osteo-sclerosis. I have made sections of twenty temporal bones in which chronic suppuration was evidenced, and eighteen of them were "infantile" in type and two were cellular. I bring them for your inspection to-day, the details being given with each. The eighteen had healed or had had persistent suppuration, and death had occurred in nine as a result. The pathway of extension from the antrum has been influenced by the anatomical condition present. The danger of the types is very evident. It will be noticed that the superior or posterior segment of the membrane is involved in all the eighteen.

Of the two cellular bones, in the first the disease of the middle ear is shut off from the antrum by a distinct cicatricial band, and there is no evidence to show that the antrum was ever affected; in the second, extensive disease of the middle and posterior fossæ is present, but I have no record of the case.

The manner in which the types are responsible for chronic suppuration from the middle-ear tract is, in my opinion, as follows: The antrum becomes infected in many cases, though not in all, from the lower middle ear in acute middle-ear inflammation, especially in scarlet fever and measles. If mastoid cells are present they also become infected, necessitating most probably the Schwartze operation. If either of the infantile types is present the infection is unable to penetrate to the mastoid process or through the outer wall of the antrum, and many things may happen:—

(1) It is possible that the drainage through the membrane is sufficient to produce a cure.

(2) Symptoms may arise necessitating the opening of the antrum. I may here say that this condition in the types cannot give rise to the classical "mastoid" signs, and I think it is important that the diploë should not be opened up.

(3) Intracranial or labyrinthine complications may be produced.

(4) Changes in, and destruction of, the lining membrane, with caries of the bony walls, and especially of the "foetal" squamous cells, or of the ossicles, may be produced and a chronic discharge from the middle-ear tract established with all its later possibilities of extension. Here also the complications take place without classical "mastoid" signs.

I should like to remark how frequently one reads in the records of operations for grave complications of chronic ear suppuration that the "mastoid was sclerosed," or words to that effect.

It must be the experience of all aural surgeons that, apart from cases of tuberculosis, they have rarely, if ever, seen a case of acute suppuration become chronic under their hands. In my opinion, this is due to the fact that the infection is most frequently due to some specific fever while the patients are isolated in a fever hospital or elsewhere, and unless an obvious complication occurs they leave their isolation with a chronically discharging ear in which an infantile type is present.

The clinical features of chronic antral implication in the infantile types are a chronic discharge from the middle ear with a perforation involving the posterior-superior or superior segments of the membrane, with or without caries of the ossicles and outer antral wall, or of granulation or cholesteatoma, with ability to draw pus from the attic and antrum by means of Peter's magnifying speculum.

I believe that the X-rays will in the future be of the greatest service in connexion with the diagnosis and treatment of suppuration in the temporal bone by enabling us to decide the type of bone present.

DISCUSSION.

Dr. PRITCHARD said he had been watching these cases, and was convinced, and had been for a long time, that Mr. Cheatle's explanation was correct. He did not think there was ever inflammatory condensation of the bones in these cases, except, perhaps, in a few cases of cholesteatoma, in which there was a little condensation just around the enlarged antrum. Otherwise, he did not think there was ever disease of the bone accounting for what Mr. Cheatle called the infantile form of mastoid.

Mr. A. L. WHITEHEAD said he had seen a case of acute otitis media, followed by meningitis and death, in which there were grounds to suspect the presence of an abscess. Therefore he operated, although the patient was very ill. The whole of the mastoid and antrum were composed of dense bone, but there could not have been any sclerosing process, as the history of otorrhœa had only extended to three or four weeks. The mastoid was an extreme example of the infantile type.

Mr. WAGGETT said he had under care a young woman suffering from chronic suppurative mastoiditis, and her mastoid proved, at the operation, to be of the infantile type. After an interval of some years the opposite mastoid was opened on account of severe and prolonged pain. Evidence of suppuration in this ear was entirely absent, and operation revealed no evidence of past or present suppuration. Nevertheless the bone proved to be of the infantile type, and this fact furnished support to the view that the anatomical condition of the suppurative ear was of developmental origin also and in no sense due to a pathological sclerotic process.

Mr. HUGH E. JONES asked Mr. Cheate to expand his remark on not exposing the diploë; did he object to operating on the diploë because it took longer to heal, or because of danger of osteomyelitis?

Mr. CHEATLE, in reply, said he would have liked to hear whether members had, as a rule, discovered the dense outer antral wall when operating for chronic suppuration. To him the recognition of the infantile types had thrown a flood of light on chronic suppuration, and many things which in his mind had been obscure were made clear. As to opening the diploë, first in regard to acute cases, in the infantile types the antrum alone contains pus and needs opening, and if in those cases the diploë were opened there was risk of infection extending into it. He recently had a case of chronic suppuration with acute symptoms, in which the antrum was full of pus. There was a dense outer wall. In opening the antrum he also opened the diploë. Three days later he had to see the patient because she was complaining of pain, and there was œdema over the tip of the mastoid. He opened it again, and found that at his operation he had infected the diploë of the mastoid, and had to clear it all out. Also in operating for chronic suppuration he thought that the diploë should not be opened if it could be avoided. In answer to Mr. West, he had stated many times that the specimens were open to inspection at his residence at any hour of the day or night, but the number of visitors who had responded to his invitation could, so far, be counted on the fingers of one hand.

Pedunculated Papilliform Growth springing from the Posterior Border of the Cartilaginous Meatus.

By W. MILLIGAN, M.D.

C. M., MALE, aged 28, came to hospital complaining of an almost continuous boring pain in his left ear of eight months' duration, which he attributed to the result of a blow. Upon examination a nœvus was found to occupy the whole of the left tragus. The auditory meatus was entirely occluded by a pedunculated papilliform growth

springing from its posterior margin and almost filling the concha. On pressing it forcibly to the side a view of the membrana tympani was obtained. There was no defect in hearing and tuning-fork tests were normal. The growth was removed and the following report of its structure received: "There is an invasion by malignant epithelial cells of the structures of the true skin—i.e., the processes run around the sebaceous glands and enclose them in a most intimate manner." Since the removal of the growth, the patient has had no pain and the surface from which the growth was removed has completely healed. No enlarged glands are to be felt. What treatment should be adopted?

A photograph and microscopic section were exhibited.

The growth was subsequently handed to Mr. J. H. Targett, who has kindly reported as follows: "This tumour has the general structure of a warty papilloma of the skin, the processes of which are infiltrated with a new growth, while the subjacent tissues are much inflamed. The new growth involves the corium and its prolongations into the papillæ, some of which are greatly thickened by it. The cutis vera, though in close contact with the growth in many places, is not affected by it. Histologically the younger parts of this neoplasm are composed of rounded alveoli filled with small cells having a clear oval nucleus. In the other parts of the tumour all trace of alveolation is lost, and the whole papilla is occupied by closely-packed cells. The alveolation, where most distinct, is formed by a single layer of flattened cells like endothelium, and apparently there is no connective-tissue proliferation in the growth. We are of opinion that this new formation is an endothelioma derived from the lymphatic vessels of the corium and papillæ, and that it is of a malignant nature, though possibly in a low degree."

DISCUSSION.

Dr. MILLIGAN said he would have liked to have heard some expression of opinion from microscopists as to whether it was a malignant growth. The report was that there was an invasion of epithelial cells. Some portions of the section seemed to present an innocent appearance, and it was a very important point to decide in reference to treatment. Complete healing had taken place, and he did not feel justified in doing anything further, though if it was malignant he would do so. Here, as was so often the case, the patient dated the onset of the growth from an injury. He had previously shown a case of epithelioma of the middle ear, where it was stated that the trouble followed a blow over the ear.

Mr. WEST said he had had a similar case, a papillated growth in the meatus, pedunculated, in which there was much doubt as to its malignancy. Section through part of it looked like innocent papilloma. Sections through the basal portions were reported upon by skilled pathologists as being almost certainly malignant. As the growth was deep in the meatus, he felt it was right to remove the whole meatus. That was two years ago, and there was no recurrence. He advised Dr. Milligan to leave the case alone at present, but watch it carefully for recurrence. If it did recur, the whole meatal wall should be removed, with the pre-auricular and mastoid glands and the tissues between *en masse*.

Dr. DUNDAS GRANT said they must not be too impressed by the microscopic data, especially when they were not absolutely characteristic. Two or three years ago he saw a lady who suffered from intense pain in the ear, and in the deeper part of whose meatus was an ulcer with everted edges. A portion of it was removed for microscopical examination, when it was said to be invaded by epithelial cells and was almost certainly malignant. He scraped it, and for some reason the pain disappeared, as also did the ulcer. Another case Dr. Mackenzie saw with him, which answered much the same description, except that it was a flatter ulcer, with fungating edges, and the report here also was that it was almost certainly epithelioma. He removed it and cauterized deeply the parts from which it arose, and no recurrence took place. But he believed that the meatus was almost completely closed. The case was probably not malignant.

Mr. MACLEOD YEARSLEY endorsed Dr. Grant's remarks about the pathological appearances of such growths. Some years ago he showed before the Otolological Society a papilloma from a woman aged 54.¹ The concha was filled by the growth, which sprang from it. It was reported to be a pure papilloma, and he removed it with the connecting cartilage. A later examination from the base was made, and the warning given that it would probably turn out to be malignant. It healed easily and there had been no recurrence.

Dr. BRONNER said that if there were recurrence, radium or the X-rays might be tried. Operation would leave a marked deformity.

Mr. C. H. FAGGE said he did not think that there was anything in the clinical history of the case or the microscopical appearance of the section to suggest malignancy. He would treat it as innocent until it was proved to be malignant. The last thing he would do would be to irritate it with X-rays, which might tend to make it malignant.

Dr. MILLIGAN replied that he would like to refer the case to the Morbid Growths Committee. Meantime he did not propose to operate until there were other symptoms. He asked whether in such a case Mr. West would be satisfied with removing the portion of the external wall of the meatus, or whether he would take away the whole auricle.

¹ *Trans. Otol. Soc. U.K., Lond., 1901, ii, p. 136.*

Mr. WEST, in reply to Dr. Milligan, said he felt reluctance in sacrificing the pinna as a whole. He thought it ought to be possible to leave most of the pinna, so as to preserve the contour of the head. It would be best to remove the whole meatus with the conchal part of the pinna. The fact that they did not recur when locally removed did not point to their not being malignant, provided the case was an early one and was completely removed.

Large Nasopharyngeal Growth in a Boy aged 12.

By W. MILLIGAN, M.D.

A. W., MALE, aged 12, was seen in consultation on account of nasal obstruction, frequent attacks of spontaneous nasal hæmorrhage, and deafness upon the left side. The nasopharynx was found occluded by a large, firm, and hard growth springing from the vault and protruding into the left nasal passage. Marked deafness upon left side, membrane much retracted, and middle ear full of a non-purulent exudation. A previous attempt had been made to remove the growth through the nasal passage, but had been abandoned on account of its impracticability and the profuse hæmorrhage which ensued. Under chloroform the left upper jaw was excised and access obtained to the nasopharynx; growth removed; uninterrupted recovery.

The specimen was exhibited.

DISCUSSION.

Dr. PATERSON said that twice he had removed a similar growth by a simple method which had been described by Brady. He used a Langenbeck's periosteum elevator through the nose, and with one finger in the nasopharynx, enucleated it. In that way he got away the whole growth, without any operation on the upper jaw or splitting the soft palate. He (Dr. Paterson) had done it twice, and in one of the cases the growth was as extensive as Dr. Milligan's. There had been no recurrence in either case, and the method was worth consideration.

Dr. FITZGERALD POWELL said that he understood from the notes that this growth had been a fibroid growing from the "basisphenoid." He certainly would not consider himself justified in advising "excision" of the jaw for the removal of a non-malignant tumour growing from the base of the skull. He (the speaker) had removed a dense fibroid as large as his fist, growing from the

base of the skull, by gagging the mouth open and splitting the soft palate, chiselling away a portion of the hard palate, and in that way getting good exposure and free access to the tumour. He showed the boy before and after the operation, with the tumour, which was very large. The patient made a good recovery, and there was no recurrence. In such a case there was no need to remove the upper jaw.

Mr. YEARSLEY said he had only met with one such tumour, in a sailor, aged 30, and he got plenty of room by splitting the soft palate.

Dr. STCLAIR THOMSON said he had examined Dr. Powell's specimen, and the circumference of it was 8 in. The patient was not disfigured. He (the speaker) had lately made a tour of the London museums, and it was striking to see the number of cases in which the jaw had been needlessly sacrificed by the general surgeon. The upper teeth and the floor of the antrum were healthy and intact in many cases. He thought it was possible to deliver most of these tumours through the mouth, or through the opening in the ascending process of the superior maxilla, reaching it by a Moure's operation, or by a combined Rouge and Moure operation.

Dr. PETERS said he had done the operation in two cases as mentioned by Dr. Powell, and it was quite satisfactory. In one of the cases he did laryngotomy, but not in the other. The hæmorrhage incidental to chiselling the central part of the hard palate was small.

Dr. DUNDAS GRANT said that one of the greatest services which rhinologists had rendered was the early detection of those tumours, and the removal of them in suitable cases through the natural passages. There were some good periosteal elevators devised by Guyon in Paris, and with them, aided by the finger in the pharynx and working partly through the pharynx and partly through the nose, one could generally get the better of those tumours. It was wise to be prepared for great hæmorrhage, though it might not occur.

Mr. FAGGE said that he had only once removed a large growth from the nasopharynx, and the chief difficulty was to get the free end of the growth over the epiglottis and the base of the tongue, as it was almost impacted in the pharynx. Passing the loop of the snare through the nose, he caught it with a finger in the mouth, and then passed it over the apex of the growth; then pushing the wire up to the base of the growth it was made tight, and the growth was easily detached and delivered through the mouth. Extreme hæmorrhage at this point had been anticipated, so a vast array of instruments, including those for tracheotomy, had been got ready, but nothing happened. With an adenoid curette, the base of the growth was scraped away. The patient was subsequently seen many times, and there was no recurrence. The only difficulty was to deliver the lower end of the growth into the mouth, because it went so far beyond the apex of the epiglottis, and was fairly thick in transverse section. He recently had occasion to study the literature of removal of nasopharyngeal growths, and found it generally agreed that the best way of getting access to the nasopharynx was by Nélaton's operation—i.e., splitting the soft palate and, if necessary, resecting

the hard palate. Another alternative to splitting the soft palate was osteoplastic resection of the upper jaw. Excision of the upper jaw was, in the opinion of most of the surgical authorities, unjustifiable, on account of the amount of deformity it produced.

Dr. MILLIGAN, in reply, said it was difficult to criticize a case such as he had mentioned without having seen it. He would be the last person to excise a jaw unnecessarily. When the patient was on the table, three alternative methods of treatment were considered—viz., removal with the snare, splitting of the soft palate, and either resection or actual removal of the upper jaw. The circumstances of the case were such that it was not only his opinion, but also of his colleagues, that the proper course of procedure in this particular case was removal of the upper jaw. He considered that many of the alternative operations suggested for the removal of post-nasal growths were absolutely inefficient, in that it was not always possible to entirely remove the growth, that occurrences were undoubtedly more frequent, and that the difficulty in arresting hæmorrhage was very much greater than when the half of the upper jaw was removed.

Demonstration of Kuhn's Instruments for Per-oral Intubation.

By W. MILLIGAN, M.D.

KUHN's instrument consists of an armour-plated and flexible tube, with a curved and rigid stilette, mounted upon a handle. The instrument is rapidly introduced into the larynx, the tongue being drawn forward and the epiglottis tilted forwards, with the left index finger introduced into the mouth. The moment the instrument is felt to have entered the larynx the stilette is withdrawn and the instrument freed by means of a rubber band passed round the neck. The instrument is made in several sizes.

DISCUSSION.

Dr. PATERSON said he had seen the instrument used in some Continental clinics, and it was very useful. There was sometimes a difficulty in putting the tube in when the larynx was very deep, but the method was worthy of use to avoid laryngotomy.

Dr. STCLAIR THOMSON asked what was the benefit of using this instrument compared with laryngotomy. The instrument seemed to block the mouth when

it was particularly required to have it free. The comfort in giving the anæsthetic through the neck was that the anæsthetist was out of the way. He did not think there was more traumatism from the cutting operation, if a soft sponge was neatly packed on to the larynx, than from wearing the tube. He had such confidence in laryngotomy that he thought one should hesitate before adopting the instrument shown.

Dr. DUNDAS GRANT said the instruments were a great improvement upon the stiffer form of tube, and he would be glad to hear what cases Dr. Milligan had used them for, as the results of actual experience were most valuable.

Mr. FAGGE asked whether Dr. Milligan could compare the process with Crile's method of giving open ether through rubber tubes passed through the nostrils into the pharynx.

Dr. MILLIGAN, in reply, said that he had first seen the instrument used when abroad and in cases of maxillary antral empyema. During the performance of the operation the patient's condition seemed quite satisfactory; there was no cough and no expectoration of blood. He procured his instruments from Windler, of Berlin, and had used them on many occasions in operations upon the frontal sinus, maxillary antrum and post-nasal fibroids, and had found no particular difficulty in passing the laryngeal tube. It was important to stand above the level of the patient and to draw the patient's tongue well forwards in order that the instrument could be rapidly passed into the larynx. By means of an elastic band passed round the neck the instrument could be kept fixed. He had only seen traumatism of the larynx in one case, and that was only a transient laryngitis. The instrument had to be passed quickly because the stilette had to be withdrawn rapidly. Once the instrument had been passed, the mouth was packed with gauze, which was removed at the end of the operation.

Case with Well-defined and Transitory Ménière's Symptoms ; ? Migraine with Auditory and Vestibular Phenomena.

By DAN MCKENZIE, M.D.

THE patient is a man, aged 57, spare in habit and somewhat nervous in disposition. When first seen he had been suffering for three or four months from attacks of vertigo and vomiting, which came on regularly every seven or eight days, and interfered with his activity to such an extent that he was compelled to give up work (post-office official). The first attack was experienced about eighteen months ago, but there was no repetition of the symptoms until the present series began. Each paroxysm passes through a definite and precise series of stages. The

first symptom noticed is that of hyperacusis, and at first it affects the left ear only. Beginning gradually, it slowly becomes more marked, extending to the right ear also, until even slight sounds become unbearable. This introductory stage occupies two or three days. On the third day vertigo sets in somewhat abruptly and rapidly gets more and more severe, until the patient is unable to keep upright. Sickness and vomiting ensue and continue for about an hour, the vertigo meantime remaining unabated. As the severity of the attack subsides the patient states that his head and scalp become very sensitive to touch, but there does not seem to be any actual headache. The vertigo is accompanied with deafness so considerable that he can only hear shouts. The paroxysm terminates in a deep sleep, lasting five or six hours, from which he awakes to find himself relieved from all discomfort, including the deafness, save that of weakness. The whole attack from beginning to end lasts from three to four days.

Hearing: He has been "slightly deaf in the left ear" since an attack of influenza twenty years ago. There is no history of discharge from the ears.

TESTS.

			Right		Left
Watch	= 2 in.	...	- ∞
Galton	4.6	...	5.6

TUNING-FORKS.

				Right		Left
64 V.D.	{	Meatus	...	-5	...	-10
		Mastoid	...	-9	...	-8
		Rinné	...	+	...	+
128 V.D.	{	Meatus	...	-6	...	-10
		Mastoid	...	-9	...	-20
		Rinné	...	+	...	+
256 V.D.	{	Meatus	...	-3	...	-10
		Mastoid	...	-2	...	-4
		Rinné	...	—	...	—
		Weber	...	>	...	>
512 V.D.	{	Meatus	...	-2	...	-6
		Mastoid	...	-4	...	-2
		Rinné	...	+	...	+

VESTIBULAR REACTIONS.

		Right		Left
Caloric (22° to 24° C.)	Nystagmus in 30 seconds	...	Nystagmus in 50 seconds	...
	Slight vertigo	...	No vertigo	...

That is, very slightly impaired response right, and considerably impaired response left; corresponding closely with the hearing powers.

General condition: No albuminuria; pupils and knee-jerks normal; fundus oculi normal; arteries rather tortuous. The patient has always been nervous and complains greatly of the worry and pressure of his occupation.

Treatment: The patient was reduced to a strictly vegetarian diet for several weeks, and was given 8 gr. to 10 gr. of potassium iodide three times a day, with immediate benefit. For, although he found the treatment "lowering," he confessed that a definite attack of vertigo did not occur during this time. In consequence of the success attendant upon these measures, a relaxation of the stringency of his regimen has been recently permitted.

The evanescent character of the symptoms puts the explanation of recurrent hæmorrhages out of court, and it is also difficult to imagine that a serous effusion would occur with such regularity and disappear with such rapidity. Consequently the exhibitor is inclined to assume a vasomotor disturbance as the most likely cause of the symptoms, possibly toxic in origin, and affecting the auditory system in a manner that may resemble the ocular disturbances of migraine. No paræsthetic or paretic phenomena were complained of, however, if we except the hyperæsthesia of the scalp. But a patient suffering from violent vertigo is naturally incapable of appreciating minor nervous disturbances.

DISCUSSION.

Dr. DUNDAS GRANT said that since the vertigo seemed marked, and it was not merely confusion, the term "Ménière's symptom" was appropriate. It seemed to be on all fours with the cases described in Politzer's text-book as angio-neurotic disturbance of the internal ear. He asked whether it was usual in the normal subject to get nystagmus and vertigo in thirty seconds. Did not that indicate that there was an increased sensitiveness of the vestibular nerve rather than otherwise?

The PRESIDENT (Dr. E. Law) asked whether Dr. McKenzie associated the transitory symptoms with the lesion which caused the slight deafness in the left ear, also whether he had tried rarefaction of air in the left meatus at the onset of the premonitory symptoms. He did not know why the patient was put upon a vegetarian diet, because such a condition seemed to be associated with neurasthenia, which required tonic treatment rather than lowering measures.

Dr. PRITCHARD desired to associate himself with the President's remark as to the diet, and said the lowering treatment so long advocated by physicians was wrong.

Dr. McKENZIE replied that he had hoped to hear some comments upon the comparison of the symptoms with the ocular phenomena of migraine. There seemed a similar state of matters here to those found in migraine, and it was for that reason vegetarianism was ordered. Members would recollect the migraine was very frequently benefited by an abstinence from butcher's meat. The result seemed to have justified the therapeutic method which had been employed, for the patient was certainly much better. With regard to the vestibular reaction, thirty seconds was about the average duration of the induction period in normal cases. He had characterized the response as "very slightly impaired" because the vertigo was so slightly marked. He did not consider the induction period to be short unless it was less than twenty seconds.

Thickening of the Cartilage of both Auricles (? Othæmatoma).

By DAN McKENZIE, M.D.

PATIENT, a florid, healthy man, aged 45, first observed his "ears" becoming "thick" about eight months ago. The pinna of both ears is the seat of hard, irregular, rounded thickenings, closely attached to and, indeed, seemingly incorporate with the cartilage. The evolution of the thickenings has been watched while the case has been under my care. A small, fluctuating, cyst-like swelling suddenly appears (? sub-perichondrial), and gradually becomes less in size and hard in substance until it assumes the aspect described.

The patient has never been insane and there is no insanity in his family. There is no history of traumatism. His occupation exposes him at times to cold, but not to any unusual extent.

According to Politzer, bilateral othæmatoma is uncommon.

DISCUSSION.

Dr. MILLIGAN asked whether there was any history of syphilis in the case, as he could not reconcile othæmatoma with the symptoms described. It seemed to be a localized perichondritis.

Mr. HUGH E. JONES said he had a case under treatment which might be mistaken for Dr. McKenzie's, so like were the respective ears. The affection of one ear was advanced and the other just beginning. Both trauma and syphilis had been excluded as far as was possible. The condition seemed to be unaffected by any kind of treatment. He considered it to be a perichondritis.

Dr. McKENZIE replied that there was no history of syphilis in the case, and that the patient had been on iodide of potassium for a considerable time without benefit. He agreed that the condition should be called sub-perichondrial effusion, but observed that in the text-books the term "othæmatoma" seemed to be applied to this condition, a title which was obviously a misnomer. He had punctured one of the swellings, and the fluid, according to Dr. Wyatt Wingrave, was purely serous, without cells or bacteria.

A Case of Audible Tinnitus.

By ALEXANDER SHARP, F.R.C.S.Ed.

A. D., MALE, aged 45, complains of a "scraping noise" in his right ear, which began about twelve years ago and has been almost constant ever since. The patient attributes it to a chill which followed a long cycle run, and he states that the character of the noise has varied very little since he first noticed it. Hearing in both ears is practically normal, and there is no marked pathological condition in the meatus, nose, or nasopharynx. Sleep is much disturbed and his general health is impaired. The murmur can be heard if the observer places his ear close to the right ear of the patient. With the otoscope the murmur is very distinctly heard and is synchronous with the pulse. Pressure applied over the carotid artery controls it.

Listening with the stethoscope it may be noted: (1) That the murmur is heard very distinctly over the right mastoid process and over the right temporal region; (2) that it is also heard distinctly over the same areas on the left side, although the patient does not hear it in the left ear; (3) that it is heard over any part of the cranium, diminishing as one approaches the middle line.

Sneezing, coughing, or inflation of the middle ear causes a momentary cessation of the sound.

DISCUSSION.

The PRESIDENT said the general opinion was that such cases were best left alone.

Dr. H. J. DAVIS asked whether the man had ever had medical treatment. He had found big doses of hydrobromic acid and nux vomica gave more relief than anything. He did not think giving iodide of potassium or salicylate of

soda did very much good. Patients with tinnitus were always depressed, and the last-named drugs only depressed them more.

Dr. DUNDAS GRANT asked whether Mr. Sharp had tried the effect of compression of the vertebral arteries at the appropriate points at the back of the neck. If so, and it gave relief, it would be well to apply little pads of cork at the spots, and put a bandage round to keep them in place.

Mr. SHARP replied that as the man had had the symptoms for twelve years he had visited many aural surgeons, and had had all forms of treatment, each doctor he had consulted winding up with the advice that he should keep up his general health and forget all about it. He thought ligature of the internal carotid might have a beneficial effect, but he hesitated because of the probability of collateral circulation being set up, and the last stage being worse than the first. The patient got great relief by pressing his finger behind the angle of the jaw, and turning his head to the right. Inflation of the middle ear caused the sound to disappear for about thirty to sixty seconds.

Mucous Polypus presenting at the Pharyngeal Orifice of the Left Eustachian Tube in a Man suffering from Bilateral Chronic Adhesive Otitis Media.

By J. ARNOLD JONES, F.R.C.S.Ed.

MALE, aged 45. History: Deafness for ten years; onset after rheumatic fever. Right ear the first to be attacked, the left following some years later. No tinnitus, no vertigo, no discharge. Paracusis Willisii present. Has been troubled slightly with nasal obstruction and discharge for about five years.

Examination: Right ear—Drum slightly retracted; malleus fixed. Left ear—Drum very retracted, especially in upper quadrants; opaque; malleus fixed. Eustachian catheter applied to right ear produces no improvement. Sounds are distant and small. Eustachian catheter applied to left ear produces marked improvement. Sounds, at first distant and sibilant, become normal.

Examination of nose by anterior rhinoscopy: The nostrils are narrow and the inferior turbinates are hypertrophied, especially the posterior ends. No signs of polypus formation.

Examination of nasopharynx by posterior rhinoscopy: Presenting at the orifice of the left Eustachian tube is easily seen a round, smooth,

greyish-blue tumour about the size of a large cherry-stone. This entirely fills the lumen of the orifice, and seems to bulge that portion of the lateral pharyngeal wall surrounding the orifice towards the middle line. This tumour has varied slightly in size at different times, and is now smaller than ever, but there is more bulging of the lateral pharyngeal wall. The posterior ends of the inferior turbinals are hypertrophic.

It will be noted that the polypus appears on the side of better hearing power. This patient has been under observation for two years, during which period his hearing power has considerably varied. At present it is better than when first seen, though not as good as after the first course of treatment. Roughly speaking, it remains stationary.

TUNING-FORK AND OTHER TESTS.

			Right		Left
Acoumeter	22 in.	...	30 in.
Voice	16 in.	...	8 feet
Whisper	Nil	...	6 in.
Rinné C.	Negative (8)	...	Negative (8)
Rinné C ₂	Just positive	...	Just positive
B.C. C on Mastoid	+ 10 sec.	...	+ 12 sec.
Galton. W.	- 1 revolution	...	- 1 revolution
Air Conduction :					
3 C (16)	Nil	...	Nil
2 C (32)	Nil	...	Nil
1 C (64)	Nil	...	Nil
C (128)...	- 30 sec.	...	- 25 sec.
C ₁ (256)	Much diminished	...	- 15 sec.
C ₂ (512)	Much diminished	...	- 8 sec.
C ₃ (1024)	Nil	...	- 18 sec.
C ₄ (2048)	Nil	...	- 20 sec.

Mr. ARNOLD JONES said the case had been under his observation two years, but there had been intervals of four and six months. When the patient first came he was catheterized for five weeks, rested for three weeks, and then he came again. The hearing had improved on the left side. He had not attempted operation, because the hearing power on that side improved, and that was the ear he depended on for hearing. He did not care to risk an operation on a comparatively sound ear. He could find no record of a similar case, and he regarded the diagnosis as being between that of a mucous polypus and a cyst. He thought puncture would do no harm, and might clear up the diagnosis.

**Osteomyelitis of the Right Temporal Bone, secondary to
Mastoid Disease ; Removal ; Recovery.**

By HUNTER TOD, F.R.C.S.

A GIRL, aged 18. Chronic middle-ear suppuration in both ears, of eight years' duration ; the sequela of scarlet fever. First seen in Out-patients' Department on February 12, 1909. In right ear, large perforation of tympanic membrane, with granulations in upper quadrant. Pain behind ear, but no tenderness on pressure, and no swelling or œdema over mastoid process. Conservative treatment caused some improvement. On March 26 not so well : pain in head, excessive tinnitus. On April 3 there was some swelling over the mastoid process, and the patient was taken into hospital for the complete mastoid operation, which was performed (in my absence) by my house surgeon. The mastoid process was extensively carious, and there was an area of necrosed bone, about 1 in. square, lying over and in front of the lateral sinus. No note was made of the condition of the tegmen tympani, nor of the squamous portion of the temporal bone. The wound cavity was left open and lightly packed. On April 15 the parotid region became swollen, and, on pressure being applied, pus oozed into the wound cavity through a fistula in the anterior meatal wall. The œdema and swelling gradually increased over the parietal and temporal region. The temperature varied between 99° F. and 101° F. for two weeks, and then became normal to subnormal. The patient was well otherwise, except for occasional headaches, sometimes acute ; but seemed to have fits of drowsiness.

On May 5 I made an incision from the upper angle of the mastoid wound and upwards over the temporal bone, and exposed the whole of the squamous portion, as well as the zygoma. The surface of the bone was pitted, worm-eaten, and hæmorrhagic in patches, and small flakes of necrosed bone could be separated off. The whole of the squamous bone was removed with forceps until healthy bone and dura mater were reached. The underlying dura mater was thick, rough, and covered with purulent secretion and granulations. The posterior half of the zygoma, the whole of the tegmen tympani, together with the anterior wall of the bony meatus, were also necrosed and removed, so that the condyle of the lower jaw could be felt. The wound surface was irrigated freely with weak biniodide solution, and packed lightly with

58 Davis: *Adventitious Membrane formed after Operation*

gauze. Convalescence was gradual, the patient not leaving the hospital till July 1, the wound being then practically healed over. A shield of aluminium was worn to protect the brain over the area deprived of its bony covering. The pulsations of the brain are still visible over the wound area, though to a much lesser extent than in July. The case is very similar to, though less extensive than, the one I communicated to the Section on February 6, 1909.

DISCUSSION.

Dr. H. J. DAVIS recommended that the shield should be dispensed with. It prevented the hair from growing over the area of bone removed, and in the case of women their hair was a great protection. A girl aged 10, a patient of his, had part of the skull removed for brain abscess following mastoid disease, and when she left the hospital two years ago, the brain pulsations over the bulging area were so marked that they were communicated to her hat when she wore one. He had seen her a few days ago, and it was surprising how hard and tight the area operated on had become. He thought that the sooner the metal shield in this case was abandoned the better.

Mr. TOD, in reply, said that the only object of putting the shield on was to act as a protection during the first few months until thickening of the overlying tissues took place, which in most cases afterwards acted as a sufficient protection.

**Adventitious Membrane, resembling normal Drum, formed
after Radical Mastoid Operation.**

By H. J. DAVIS, M.B.

GIRL, aged 15. A radical mastoid was performed in 1908. The cavity was dry and healed in five weeks. Three months ago she came to the hospital with "earache following a cold in the head," and mucus could be detected behind what was taken to be the drum. This is evidently not so. The Eustachian tube was curetted at the time of operation and the remains of the drum removed. An adventitious membrane has formed which so closely resembles a normal drum that its real nature might be overlooked. The membrane, which is post-operative scar-tissue, is very thin and translucent, and insensitive to the probe. The opening into the antrum can be seen in a plane nearer to the observer. The hearing power is remarkably good.

DISCUSSION.

Mr. HUNTER TOD agreed with Dr. Davis in that he had seen cases in which the hearing power had been remarkably good after an adventitious membrane had formed, which apparently acted as a drum. He had one case in particular where the patient's hearing seemed normal. The formation of this adventitious membrane was, however, an accidental occurrence.

Dr. FITZGERALD POWELL said he thought there must be some mistake about the improvement in the hearing, as he thought the hearing was lost in that ear. The covering was excellent and the ear dry; it was a good result, but the hearing was not good.

Dr. KELSON said he showed a case of the kind before the Otolological Society. In his case an adventitious membrane had formed, and that patient could hear the watch at 12 in., the normal distance being 30 in. If, as Mr. Hunter Tod had said, the hearing might be practically normal in such cases, it was interesting as showing that the ossicles were unnecessary for the transmission of sound.

Mr. HUGH E. JONES suggested that the good hearing in this case was due to moisture in the tympanum. He always found that if the mucous lining of the inner wall of the tympanum was retained, the stapes acted better and the moisture produced by the mucus secreted aided in the hearing. Where the tympanum was absolutely dry, hearing was not so good as where it was moist.

Dr. FITZGERALD POWELL said that hearing depended very much on the condition of the labyrinth, and whether injury was done to the inner wall of the tympanum at the operation. If the inner wall or the stapes was interfered with the hearing would be bad.

Dr. MILLIGAN said that he agreed there were cases in which an adventitious membrane, which formed close to the situation of the original tympanic membrane, assisted audition. Two very important factors had to be considered *re* the question of the preservation of hearing—viz. the integrity of the inner wall of the middle ear, and the integrity of the stapedio-vestibular joint. He regarded the membrana tympani as of more use to prevent evaporation from the middle ear than actually to convey sound. When the tympanic mucosa became dry, hearing suffered. When, on the other hand, it remained in a moist condition, the chances of improved hearing were distinctly greater.

Dr. DAVIS replied that the hearing was previously excellent; he had repeatedly shown her during the last two years as an example of this. Since she had had this attack of earache, she said she had not heard so well, but she would recover from this. If one made her perform Valsalva's experiment, the membrane could be seen to bulge. He thought that failure to occlude the Eustachian tube at the time of operation by curetting was the explanation of this patient's symptoms. The tube has been curetted, but evidently not obliterated. In a radical mastoid there was no object in still leaving a communication into the nose, and recurrence, too, of discharge might occur with successive colds, and this often happened after an otherwise well-done operation.

? Papilloma on the Posterior Superior Quadrant of the Right Drum.

By H. J. DAVIS, M.B.

THE patient is a girl aged 10. The appearance of this little growth is peculiar. There are no symptoms referable to the ear, and it was merely detected in the course of a routine examination. The child was attending for follicular tonsillitis, and the tonsils have now been removed.

Left Facial Paralysis following a mild Catarrh of the Middle Ear. No Perforation.

By H. J. DAVIS, M.B.

THE patient shown, a woman aged 45, is one of two women with a precisely similar condition attending the hospital at the present moment. In both cases complete facial paralysis resulted from a mild attack of earache following coryza (? influenzal). In the patient exhibited this occurred five months ago, and she is recovering very slowly under electrical treatment. When first seen the membrane was indrawn and there was slight mastoid pain, but there was no redness of the drum. The local conditions rapidly subsided, but the paralysis remains and still persists. Hearing is normal. She states that many years ago she had slight earache on the other side, and "the face was drawn over to the left for months." It has been suggested that this is a neurosis, but the reaction of degeneration is present in the facial muscles, and the condition is probably due to some abnormality or deficiency in the canal of the facial nerve.

DISCUSSION.

Dr. DUNDAS GRANT said there was probably some dehiscence in the canal. She lost the sense of taste on the left side of the tongue for some time, and she still had extreme sensitiveness to sudden noises on the paralysed side, indicating paralysis of the stapedius, and loss of the protective influence of that muscle.

Dr. DAVIS, in reply, said that whenever the facial nerve was paralysed or paretic from whatever cause, it took a long time to get well. Some cases of Bell's palsy following colds, &c., never recovered at all, in spite of electrical treatment continued for years.

Case of ? Congenital Defect of both Tympanic Plates.

By C. ERNEST WEST, F.R.C.S.

FEMALE, adult. When first seen, patient had a long-standing bilateral otorrhœa; on the left side there was a scar over the mastoid, and the meatus was greatly contracted. After a radical mastoid operation the meatus again became almost closed, with continued suppuration. Recent creation of left mastoid fistula and right radical mastoid operation. The right tympanic plate appeared to exist only in its deepest part; the posterior wall of the bony meatus was formed by the anterior surface of the mastoid; there seemed to be bone in the deepest part of the floor and anterior wall, but for the most part both were membranous. There was no feature suggesting that destruction of bone had taken place; the meatus on this side was roomy and of usual form. On movement of the jaw the excursion of the condyle is still clearly visible. The condition suggests a failure of development of the tympanic ring into the tympanic plate and the retention of the infantile form.

Case of Endothelioma of Temporal Bone.

By D. R. PATERSON, M.D.

THE patient, a woman aged 27, was admitted to hospital on December 31 last. She gave a history of offensive discharge from the left ear for at least fifteen years. This continued without change until, two months before admission and one week after her second confinement, she was seized with severe pain in that ear, and this had persisted without remission. She looked extremely ill, but was quite sensible and gave a clear account of her illness. The temperature was 99.2° F., and pulse 92. The left auricle and surrounding parts stood out from the side of the head and greyish-yellow offensive discharge oozed from the meatus. There was bogginess behind, above, and in front of the auricle, with slight pitting on pressure. There had been no hearing in the left ear for years, and the patient's state did not permit of definite tests. Slight nystagmus was noted on looking to the right, and the optic disks were normal. An operation was performed the same day. The usual

posterior incision through oedematous tissue came upon a large cavity filled with soft greyish masses occupying the site of the antrum and the mastoid cells. The posterior wall of the meatus had disappeared completely. There was very little pus. At first sight it looked as if the bone were scooped out by old-standing cholesteatoma, but the soft friable masses gave rise to suspicion of new growth. The soft tissue was easily removed without much bleeding, and it was seen that the temporal bone had been hollowed out to an extensive degree, laying bare a large part of the dura, both of the posterior as well as the anterior fossa. The roof of the antrum and tympanum and a part of the squama had disappeared, and the ragged bone showed signs of infiltration by growth. Wiping the tympanic cavity produced twitching of the face, and the prominence of the semicircular canal stood out well. The removal of only very little superficial bone was required to complete the exposure of the cavity, and trimming the bony edges of the gap in the squama appeared to get rid of all affected bone. A smooth cavity of large size was left, and this was packed with gauze. Microscopically the growth appeared to be an endothelioma.

The patient was much relieved by the operation. After a few days the growth began to appear again, especially in the upper part of the cavity. A rise of temperature began on January 19, and the patient became dull and listless, whilst the boggy condition appeared above the auricle. On January 26 the cavity was cleared of fresh growth, which had extended further along the squama towards the root of the zygoma. The exposed middle fossa dura appeared slightly thickened, but, as the growth peeled off readily, it was not further explored. The patient died four days later.

Summary of post-mortem report by Dr. Schölberg: There was localised basal meningitis and the temporo-sphenoidal lobe was adherent to the dura at a point over the left petrous bone. Microscopic examination of the dura and adjacent part of the brain showed the growth to be an endothelioma which had grown from without through the dura into the brain substance.

The temporal bone and microscopic sections were exhibited.

Dr. PATERSON added that the pathologist reported that the section was endothelioma, at the same time noting that there were certain appearances which resembled a rapidly-growing carcinoma, though the clinical history was obviously different from that. He would like the Morbid Growths Committee to report upon it. Dr. Grant's similar case was sent to it, and it might be well to compare the two.

Otological Section.

June 4, 1910.

Dr. EDWARD LAW, President of the Section, in the Chair.

Notes on the Pathogeny of Cholesteatomata.

By V. H. WYATT WINGRAVE, M.D.

CHOLESTEATOMATA occurring in the petro-mastoid bone are of several varieties: (1) Those which are encapsuled or enclosed in a definite sac; (2) those which are diffuse, or possess no defined sac or wall, being generally associated with granulomatous and inflammatory changes; (3) those of a vestigial type or which originate in cell inclusions; (4) those which are believed to be composed of endothelial elements. The first and second type have chiefly been the subject of observation, and it is their probable pathogeny that will be considered in these notes.

The first, or encapsuled, type is seen as a pearl-like sac completely filling a space in the petro-mastoid, belonging either to the antro-tympanic chamber or its adjacent pneumatic cavities, from which it is sometimes easily removed. These cavities are, in their normal state, lined by a single layer of short columnar or cubical epithelium resting upon a thin fibro-vascular endosteum. The sac of the cholesteatoma consists of a somewhat thickened fibro-vascular sheath containing a few plasma cells and lined by stratified epithelium, similar to the Malpighian layer of the epidermis, consisting of columnar, spheroidal, and polyhedral cells in successive layers from below upwards. The mimicry is still further emphasized by the presence of eleidine granules in the layer which corresponds with the *stratum granulosum*, by prickles and by the formation of papillæ. The horny layer is represented by the sac contents of closely-packed and laminated acid-fast squames, either devoid of nuclei or simply showing their faint outlines. Between these

cells are sometimes found amorphous fatty granules and rhombic crystals of cholesterin. This transformation constitutes a true metaplasia. How is it brought about?

The normal tympanic membrane externally is one of the driest structures in the body. Its meatal aspect is epidermal in structure, with a slightly greasy tendency, due either to fatty changes in its own epithelium or to the influence of adjacent ceruminous glands. Internally, however, it is moist and covered by a single layer of short or flattened

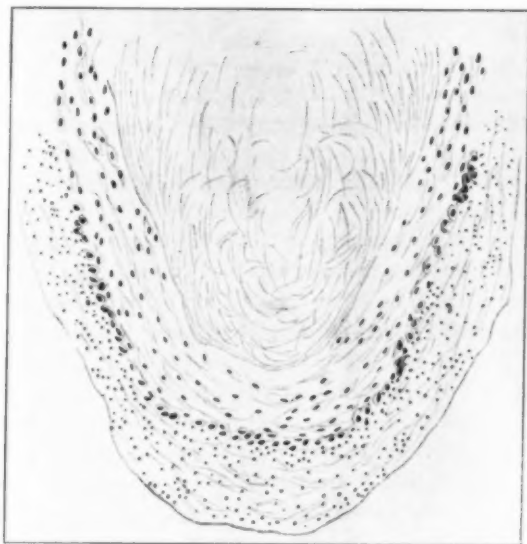


FIG. 1.

Capsule of cholesteatoma (obj. $\frac{1}{4}$ in.).

cells, the rest of the tympanum containing either cubical or columnar epithelium, which in a few places is ciliated. The scarcely perceptible moisture is, except in morbid states, not due to flooding by any visible fluid, but is probably maintained by the high aqueous tension of the air enclosed in the tympanic cavity, the water being chiefly derived by exosmosis from the richly vascular muco-periosteum at body temperature rather than from secretion of mucous or albuminous glands, which are very scanty in the tympanum.

The average temperature of the meatus observed in healthy patients taken under constant condition was 93°F. , having a range of from 90°F. to 96°F. It may therefore reasonably be assumed that the tympanic temperature is normally at least three degrees higher than the meatal temperature, and consequently admits of a relatively higher degree of saturation. The presence of a perforation permitting the entrance of



FIG. 2.

Capsule of cholesteatoma (obj. $\frac{1}{8}$ in.). A, squames; B, Malpighian layer;
C, fibro-vascular layer.

air will not only cause a fall in the mean temperature of the tympanum, but also a diminution of the vapour tension favourable to desiccation.

Observation of discharges, curettings, swabs, and other morbid material, such as granulations, polypi, and neoplasms from the middle ear, together with post-mortem search, all prove that the normal epithelium is invariably replaced by squames after prolonged persistence

of perforations. The metaplasia is not confined to the tympanic cavity, but involves its adjacent pneumatic spaces, and their contents, such as granulomata and polypi. This new squamous epithelium, judging by comparison with cutaneous activity, is probably produced more rapidly and more abundantly than the normal columnar is, and there can be but little doubt that its cytolysis and disposal are also much more difficult: it therefore tends to accumulate, and a cholesteatoma results. Such a change is not peculiar to the ear, for identical conditions occur in the pharynx, larynx, nose and its accessory sinuses, whenever they are abnormally exposed to atmospheric influences. Nasal polypi are often seen to be covered with epithelium on their exposed surfaces, normal ciliated cells being found on the protected and deeper portions. A chronic inverted uterus, or a prolapsed rectum, shows similar metaplastic changes.

Cholesteatomatous formation may, however, be influenced by other factors, such as the irritation of bacteria, toxins, tryptolysis, dust, &c. Powell White [1] has recently shown that epithelial metaplasia can be produced by injecting fatty acids. Since these have already been shown [2] to be responsible for acid-fast properties in bacteria, &c., and that these acids are ever present in chronic suppuration of the middle ear, it is not at all unlikely that they should be important factors in cholesteatomatous changes. Fortunately a large proportion of cholesteatomata are sterile, nor is there any evidence of past or present inflammatory processes in their sacs. But a simple or non-inflammatory cholesteatoma is always liable to infection, and so become the seat of activity.

The process of desiccation may also be due to changes in the quality and quantity of the local secretions, for, although the antrum and its adjacent spaces are deficient in glands, the epithelium itself may be secretory in function.

Occasionally bacteria of the mycelial type are found among the squames, but their saprophytic reputation must be regarded with some suspicion in such a situation.

The second, or granulomatous, variety of cholesteatoma is very different in appearance and structure. It is rarely encapsuled, and is generally fragmented when removed by operation, being mixed with granulation tissue, blood, and bone spicules, so that its actual nature may sometimes be only established by the microscope. Granulomatous elements covered with stratified epithelium will be found embedded in loose lymphocytes, leucocytes, myelocytes, and squames in all stages of

fatty and granular changes. The squames may be loose or grouped in closely laminated "pearls," having for the most part lost their nuclei and become strongly acid-fast. Bacteria of all kinds are mixed with the cells, such as staphylococci, streptococci, streptothrices, diplococci, and the characteristic throat organisms—spirochaetes, fusiform bacilli, yeasts, leptothrices, cladothrices, &c. Several forms of acid-fast bacilli, including tubercle, are often seen. In this type of cholesteatoma there is the additional irritation of bacteria, toxins, fatty acids, and other tryptolytic products of cell destruction.

The caseous residue of old suppuration closely resembles cholesteatomatous material on removal, but it will be found on examination to be entirely free from squames, and affords no evidence of any epithelial sac. This type is often tuberculous and associated with caries.

The only available examples of the vestigial or "inclusion" type occurred in the auricle in the form of cholesteatomatous cysts embedded in the cartilage of the helix, quite unlike sebaceous cysts.

Cholesteatomata originating by metaplasia of endothelial cells, apart from their occurrence in adjacent brain and meningeal tumours, were but rarely found. As in tonsillar and adenoid tissue, their histological transition was observed in a few granulomata, probably of tuberculous origin. Their usual tendency, however, is to caseous degeneration.

It is suggested that cholesteatomata are primarily caused by the prolonged admission of air to the antro-tympanic cavity, which by lowering its aqueous tension causes desiccation and so gives rise to a true metaplasia of its lining epithelium, and that the irritation of bacteria, toxins, and cytolytic products are supplemental factors.

The following specimens were shown :—

- (1) Cholesteatoma sac with contents.
- (2) Acid-fast squames in discharge.
- (3) Mycelial infection.
- (4) Granulomatous cholesteatomata.

Metaplastic changes in maxillary antrum.

Cholesteatomatous changes in tonsils.

Cystic cholesteatoma of auricle.

REFERENCES.

- [1] WHITE, POWELL. *Brit. Med. Journ.*, 1910, i, p. 813.
- [2] WINGRAVE, WYATT. *Proc. Roy. Soc. Med.*, 1908, i (Otol. Sect.), p. 95.

DISCUSSION.

Dr. DUNDAS GRANT asked whether the author did not think that variety No. 2 was an earlier stage of No. 1; that it was only in course of time that the epithelial layer on the surface of the granulations developed and extended sufficiently to line the whole cavity? He thought that was probably the course of events; and if sections could be made of cases which had lasted some time, it would be found to be so. Dr. Wingrave's suggestions concerning the development of cholesteatoma were at all events very ingenious, and probably all the factors he had mentioned took a share in the production. Most would agree that there was also an extension of epithelium from the external surface of the tympanic membrane into the granular surface of the diseased cavity, similar to what took place under analogous circumstances in other parts of the body.

Dr. MILLIGAN said he thought this work of Dr. Wingrave was of a particularly high order, and that he had thrown light on the histogenesis of cholesteatoma. He thought the theory of the travelling of epithelium towards the middle ear had been exaggerated in their production. Cholesteatomata were comparatively common, yet how many times had one seen the ingrowth of epithelium through a marginal perforation? It was not necessary to have a marginal perforation for a cholesteatoma to occur. He had specimens showing cholesteatomata which had developed where there had been an almost central perforation in the membrane. He thought the middle ear itself was responsible for the production of cholesteatoma, and not the external auditory meatus. One had to deal with altered factors—perforation, diminution of tension, and an inflammatory process. Dr. Wingrave said the same metaplasia took place in other organs which were exposed to altered atmospheric influences—e.g., prolapse of the rectum, nasal polypus, &c., where the first thing one saw was the metaplastic process which was altering the original structure of the epithelium. Given those altered circumstances in the middle ear, in the presence of diminished tension and increase of temperature, it seemed to him that there was every factor which was necessary for the production of that metaplastic process, and he thought cholesteatoma was a middle-ear production, possibly helped by accessory ingrowth of epithelium from the auditory meatus. One must sharply distinguish between fetal "rests" and the cholesteatoma which was the result of infection. Dr. Wingrave had pointed out the important fact that there was a cholesteatoma which was sterile and one which was non-sterile. There was also the cholesteatoma which was active and that which was not. That could only be settled by microscopical examination. If the washings from the middle ear and the attic were stained and examined, and the acid-fast squames found to contain demonstrable nuclei, that was an indication for immediate interference. If the squames were

found to be without nuclei, such a case might be amenable to local treatment. He had regarded the capsule as a product of the endosteum, and as a result of the disease. The theory put forward in the paper was new to many, but it was worthy of consideration, and Dr. Wingrave was to be congratulated on his work.

Dr. PRITCHARD desired to associate himself with what Dr. Milligan said with regard to the production of cholesteatoma. He had always thought that cholesteatoma in the middle ear was much like desquamation in the outer ear. If it could only be conceded that the mucous membrane of the tympanic cavity had taken on an epidermic character, it could be thoroughly explained; and Dr. Wingrave had considerably helped him in regarding that as the chief cause.

Mr. SCOTT said the present was an opportunity for members to express their views as to the formation of cholesteatoma. Although Dr. Grant agreed with other authorities, including Bezold, who regarded the process as being due to an ingrowth of meatal epithelium, he (Mr. Scott) ventured to take the view that there were two essential factors: (1) metaplasia, which sufficed to explain why cholesteatomata were often met with in parts of the mastoid far removed from the external meatus; (2) the production of new layers of cells more rapidly than the squames from the preceding cells were removed; a narrow exit favoured this accumulation, and it was striking to find that cholesteatoma practically always originated in cavities where the exit was too narrow for the products of growth to escape freely. More accurate knowledge, such as that furnished by Dr. Wingrave, concerning the pathology of cholesteatoma would probably modify existing opinions regarding the treatment.

Mr. WEST said the paper was an important contribution to accurate understanding of the subject. One obvious instance of metaplasia was the outer end of the ordinary aural polypus, which was covered by mucous membrane, and which, in its exposed parts, was often covered by metaplastic squamous epithelium. With regard to observational distinction between the two causes which were alleged, he did not think that was possible. Whether the production of the squamous epithelium in the cavity was due to the proliferation of squames from outside, or due to a metaplastic change consequent on lower aqueous tension, the progress of the condition would be the same. Near the canal where the aqueous tension was less, or where the new growth inwards was oldest, the change would be relatively advanced, and less obvious the further one passed from the perforation. There were some features in cholesteatoma of the antrum which he found it difficult to explain on the neoplastic hypothesis. One might see a track of cholesteatoma surface extending through the aditus, with bare bone or granulation tissue on either side, forming a tongue connecting the attic with a large cholesteatoma in the antrum. He could not see why in every case of obvious cholesteatoma in the antrum there should not be obvious cholesteatoma of the upper part of the tympanum and attic and the whole

aditus; but he did not think that was a fact as a matter of observation. With regard to the microscopical observation of the squamous plaques washed out of the tympanum, he believed the presence or absence of a visible nucleus was dependent on the age of the condition. The older and better marked the cholesteatoma the less nuclear evidence in the squames. He wished to associate himself with the expression of gratitude to Dr. Wingrave for his paper.

Dr. DAN MCKENZIE said that in connexion with cholesteatoma it was authoritatively stated that the bone atrophy and enlargement of cavities in the disease were the mechanical result of pressure of the mass of the cholesteatoma; but an inspection of the bone when the cholesteatomatous membrane was removed showed it to be studded with isolated shallow pits, a phenomenon that pointed to some cause other than massive pressure, as that would induce a uniform atrophy. Turning to Dr. Wingrave's paper, the speaker said that the apparent invasion of epidermis through a perforation could be explained on his theory. Cholesteatoma was said to be more common when the perforation was near the margin of the membrane than when it was in the centre; but perforations at the margin were generally larger than those in the centre, and consequently favoured evaporation and desiccation of the tympanic epithelium. Attic perforations, which were also credited with leading to the formation of cholesteatoma, similarly favoured the same epithelial changes, for the epithelium of the attic and ossicles, lying as it did near to the perforation, was then liable to early desiccation and metaplasia.

Dr. WINGRAVE, in reply, said that Dr. Grant's and Dr. Milligan's suggestion that the formation of cholesteatoma was due to creeping in of the epithelium was a very fascinating theory, but he did not think it entirely explained all the forms, especially the first or encapsulated. There, as Mr. Scott mentioned, the distance from the meatus was so great that one must look upon the metaplastic changes in the antrum as something quite apart from a simple extension by continuity from the meatus. Another point against that fact was that one so often found cholesteatoma without any infection, a condition which was somewhat surprising if the encroachment of the meatal epithelium were entirely responsible. Therefore he thought it more likely that an entrance of air was the primary cause of the metaplasia. Sometimes air-borne bacteria were found, such as mycelia, without any other forms. Therefore his view was that a primary cholesteatoma was entirely due to a transformation of the epithelium in the cavity, brought about chiefly by atmospheric influences.

**Deformity of both Pinnæ resulting from Perichondritis
following Double Mastoid Operation.**

By H. J. DAVIS, M.B.

C. B., AGED 25, was admitted into the hospital four months ago for severe vertigo and double otorrhœa of twenty years' duration. He was so ill that he could hardly speak, and he was dull, sick, and very giddy. Double radical mastoid forthwith with immediate improvement of symptoms. Three weeks later, when up and about the ward, the right pinna became œdematous, and this was followed five days later by a similar condition on the other side. In spite of careful treatment both auricles continued to swell till they reached an enormous size, and their protrusion from each side of the head gave the patient a ludicrous appearance (he was familiarly known among the other inmates of the ward as "The African Elephant," though he was unconscious of the reason). Suppuration set in, the pinnæ were incised, and tubes and gauze drains inserted in all directions. This was done five times under general anæsthesia before resolution set in. He has just returned, after a four weeks' stay in a convalescent home, in the present condition, the auricles being inverted, puckered, and shrivelled up to the size of a baby's. No necrosis of the cartilage occurred, though the segment was stripped from the cartilage and bathed in pus. The upper half only of the pinna is affected, and the affection is symmetrical; this is due to the fact that the cartilage of the pinna consists of a single piece.

It is suggested that a gold wire, suitably curved, subcutaneously inserted in the outer rim of the helix would rectify the deformity and improve the appearance.

DISCUSSION.

Mr. A. CHEATLE asked whether Dr. Davis put anything through the cartilage. He had never had such a case, but most of the cases he had seen were due to passing a suture through the cartilage.

Dr. W. MILLIGAN asked whether any bacteriological examination was made at the time, and, if so, what organism was found. He had had a bad case following unilateral mastoid operation, and the offending organism was the

Bacillus pyocyaneus—a common cause. Considering what was implied in the mastoid operation, the wonder was that septic perichondritis was so rare. He had seen the statement that when a large flap was made the tendency to perichondritis was greater than when a small flap was made, possibly only because of the exposure of a larger surface.

Mr. WEST said he had had at hospital a case of pyocyaneus infection in perichondritis of that sort. There was no large incision into the concha, and no suture through the cartilage, but the cartilage necrosed. The account of the present case said no necrosis of the cartilage occurred yet the pinna was shrivelled up. What became of the cartilage? It had been said that pyocyaneus infection was characteristic of perichondritis of the auricle.

Mr. HUNTER TOD said that he had seen two such cases. In one case the mastoid operation had been performed and the meatal cartilage had been cut. In that case the *Bacillus pyocyaneus* was not found. In the other case the patient apparently had a furuncle of the external auditory canal, which was curetted by his house surgeon. A few days later swelling of the auricle occurred, with subsequent perichondritis.

Dr. DUNDAS GRANT said that experiments with regard to the pyocyaneus had been made by Lermoyez, of Paris, and that investigator showed at the Congress some rabbits in which perichondritis had been induced by inoculation with pyocyaneus. He also said that an absolute cure for the destructive agent was afforded by dressing with the salts of silver. He (Dr. Grant) had seen perichondritis on two occasions, and it was difficult to say what was the reason for it taking place in those persons rather than in others in whom similar conditions obtained. He thought the structure of the cartilage varied in different people. In very old people the cartilage was in nodules, separated by fibrous tissue, and the cases in which perichondritis had occurred were probably those in which the cartilage was but sparsely supplied, approximately to the condition normal in old age.

Dr. DAVIS, in reply to Mr. Cheatle, said he did pass a suture through the cartilage on the left side, because it kept tearing out. The organism was the *Bacillus pyocyaneus*. There seemed to be no signs of anything wrong until three weeks after the operation. No cartilage was removed. The ears were now smaller than when he first showed the patient, but he was satisfied, as he stated that he heard very well. It had been suggested that a curved wire might be inserted subcutaneously to rectify the deformity, but he thought there was little to be gained by this now.

**A Case of Temporo-sphenoidal Abscess in which Vomiting
was entirely Absent.**

By A. L. WHITEHEAD, B.S.

A. G., AGED 19; occasional intermittent bilateral discharge from childhood, more profuse during the last few weeks on the left side. For about a week has had severe headache on the left side of the occiput; no vomiting; no rigors; no vertigo; no constipation.

On admission: Right—anterior perforation on membrana tympani with trace of discharge on canal; left—considerable amount of pus; tympanic membrane could not be seen owing to swelling of the upper and posterior walls. No inco-ordination or paralyses of limbs; knee-jerks present and equal; no vertigo; no nystagmus. Pupils equal and react; no optic neuritis. Looks ill; answers questions intelligently, but rather slowly. Temperature, 101.4° F.; pulse, 68; respiration, 26. Radical mastoid operation performed on the left side the same day. Very extensive disease was present in the antrum and mastoid process extending back to the lateral sinus, the surface of which was healthy. The removal of the carious tegmen exposed an extra-dural abscess above, the dura mater over a considerable area being covered with granulations. Great relief followed this operation; the headache entirely passed off, and he expressed himself as feeling perfectly well, the only unfavourable sign being some irregular elevation of temperature, the pulse-rate remaining about normal.

On the fifth day after operation he did not seem so well, was rather restless, and complained of occipital headache; on the evening of that day slight difficulty in naming objects was first noticed. On the following day this difficulty was more evident, although the patient's general intelligence was quite acute and his replies brisk; the apathy noticed before the former operation had quite disappeared. The exposed dura mater over the temporo-sphenoidal lobe was incised, and a large quantity of extremely offensive pus evacuated, the organisms proving to be an almost pure culture of *Bacillus coli communis*. The abscess cavity was so large that the walls could not be reached by the top of the little finger introduced through the opening in the dura mater.

Large quantities of necrosed brain-tissue and pus were discharged for nearly three weeks after the operation, after which the cavity began

to close up. The amnesia had quite passed off by the fifth day after the operation, and the subsequent recovery was uneventful.

The interesting features of this case are the entire absence of vomiting, the occipital site of the headache, the abscess being in the temporo-sphenoidal region, and the relation between the pulse and temperature after the first operation.

DISCUSSION.

Dr. MILLIGAN said he had lately operated upon nine abscesses of the brain, six of which were temporo-sphenoidal and three cerebellar. In one of the cases, a male, no vomiting was recorded. He asked Mr. Whitehead whether the brain abscess in his case was mainly cortical or sub-cortical, or a mixture of both. He thought the cases in which the brain abscess was in the cortex were those in which one was more likely to find disturbing symptoms, such as vomiting. The cortex contained far more blood-vessels and more fibrous and cellular tissue than the white substance of the brain, and in the cases where the abscess was mainly cortical the tension was greater, and sometimes more acute and severe, than where the abscess had passed through the cortex and was diffusing into the white substance of the brain. He asked whether Mr. Whitehead could support that idea.

Mr. WHITEHEAD, in reply, said he thought the abscess was chiefly cortical originally, and extended subcortically afterwards. It destroyed so much tissue that there was no cortical layer remaining. It was the largest abscess he had opened. It was the first brain abscess he had operated upon or seen in which vomiting was entirely absent, yet a fortnight later he saw another case in which that symptom was also absent.

Hysterical Deafness with Active Vestibular Reactions.

By P. H. ABERCROMBIE, M.D., and DAN MCKENZIE, M.D.

AT a former meeting of this Section one of us (Dr. McKenzie) expressed the anticipation that the vestibular tests might, in some cases, prove to be of value in the diagnosis between hysterical and organic deafness. But no case actually bearing out this anticipation has been forthcoming until now. In other cases of apparent functional deafness examined and tested, those belonging to the neurasthenic class showed a marked shortening of the induction period of the caloric reaction, while

in those suffering from what seemed to be hysterical deafness, the reaction to the vestibular tests was found to be impaired in harmony with the amount of deafness present, thus following the usual rule.

The case now being reported is that of a girl aged 16. The diagnosis of hysteria, leaving on one side for the moment the activity of the vestibular reactions, rests upon the following facts: The onset of deafness was preceded by an attack of transitory aphonia; there is complete loss of hearing, by aerial and bone conduction, to all tuning-forks and to Galton's whistle, and yet the patient can hear the conversational voice about 3 in. or 4 in. from the ear; the degree of deafness varies considerably from time to time; there is diminished sensibility on the left side of the face and body.

The following are the results of testing the vestibular system:—

Caloric (cold water, 22° to 24° C., the period of induction being measured in seconds): Right—nystagmus in twenty-five seconds, vertigo; Left—nystagmus in twenty seconds, vertigo. Rotation: To right, normal nystagmus and vertigo; to left, normal nystagmus and vertigo.

In health the induction period of caloric nystagmus, estimated according to the above method, is from twenty to forty seconds, the average running about twenty-eight seconds; consequently in this case the vestibular reactions are fully up to the normal. This result is of special interest, since in high grades of deafness from organic disease the vestibular response is impaired pretty much in proportion to the amount of loss of hearing. Thus a normal or excessive response to the vestibular tests in cases of severe deafness, especially of long standing, may be regarded as highly suggestive of hysteria.

DISCUSSION.

The PRESIDENT (Dr. Law) said he thought the vestibular reactions might be, at times, of the greatest assistance in differential diagnosis, particularly in monosymptomatic cases. Such cases of hysterical deafness were often very difficult to diagnose, and only recognized after the cure.

Mr. SCOTT asked whether the patient was now better than she was a month ago, and what was the prognosis.

Dr. DAN MCKENZIE, in reply, said he had reported the case in order to show that with clearly-marked hysterical deafness there might be active vestibular reactions. If the case had been monosymptomatic, it might have been open to criticism as a demonstration of the vestibular reactions in

hysteria. The presence or absence of vestibular reactions depended, of course, upon the situation and extent of the nerve-block. In hysterical hemi-anesthesia, for example, with deafness, one would be surprised to find the vestibular reactions positive in the affected ear. But hysterical cases should be tested and the results reported, as it was only by the investigation of a large number that a general rule could be arrived at. If it turned out that the vestibular reactions were normal in hysterical deafness, then we had in the tests a valuable addition to our methods of diagnosis. In reply to Mr. Scott, he was inclined to think that in this particular case the prognosis was good.

Exostosis of Right External Meatus in a Boy aged 10.

By HUNTER TOD, F.R.C.S.

THE growth is pedunculated, and arises from the floor of the auditory canal. Its surface is ulcerated, and there is much purulent secretion. The tympanic membrane can be partially seen, and is intact. The hearing is normal. There is a history of a polypus having been removed from the ear two years ago, when the discharge temporarily ceased.

The question is—What is the best method of treatment?

DISCUSSION.

Mr. HAROLD MOLE (Bristol) suggested that Mr. Tod should remove the exostosis through the meatus with a small gouge, or with a curette, if soft enough.

Mr. WEST asked whether Mr. Tod had satisfied himself that the membrane was intact. Recently he (Mr. West) had a similar case, except that there was no discharge, with just such a pedunculated exostosis, in a woman, and the meatus was so blocked that no part of the membrane could be seen, nor could a probe be passed round or beyond the growth. The growth snapped off, and was lifted out with a curette, and the patient went back with a normal meatus and membrane. There were cases in which the meatus was so blocked that the edge of a visiting card could not be inserted anywhere. One was bound to operate in such cases. He had once been forced to remove a compact sessile exostosis, and asked what was the youngest patient whom Mr. Tod had seen with a marked sessile exostosis in the meatus. He had seen one in a child aged 12.

Mr. WHITEHEAD said the case raised an important point as to treatment. The treatment of the pedunculated ones was delightfully simple. Cases with a broad base were different, and he thought they were not common, at least in Yorkshire. In them one had deliberately to chisel and carve out a new meatus. It might be necessary to use a drill. He asked whether it was ever necessary to operate in the absence of suppuration in the middle ear.

Dr. PRITCHARD said that in such cases the ordinary dental stump forceps removed the pedunculated exostosis very nicely when it was only small. When the meatus was completely filled by the growth it was more difficult. With regard to the apparent rarity of multiple sessile exostoses in Yorkshire, at the Congress of 1881 two Parisians spoke on the question, one saying he saw them constantly, and the other that he rarely saw them. He (Dr. Pritchard) believed, as he said at that time, that the explanation was that one of the doctors had a large private practice, while the work of the other was chiefly among hospital patients. He believed that the more favourably circumstanced people suffered from exostoses more frequently than the labouring class, because gout and the morning tub were the chief causes of these growths. In the present case he did not think the hearing was affected, and therefore the middle ear was probably intact. He doubted whether it was a true polypus that was removed some time ago. The pedunculated exostoses were different, histologically, from the multiple; on the outside they were soft and not tender to touch. On making a section the ossification was seen to be proceeding from the centre. The other exostoses were very hard, with a very thin layer of skin over them, and were tender to the touch, though on removing the skin they were insensitive in the middle, so that he had drilled them without an anæsthetic at a second operation when the outer part had been removed at the first.

Dr. MILLIGAN questioned whether it was correct to call it an exostosis. He believed it to be a hyperostosis. He agreed with what Mr. Whitehead had said: in the North of England exostoses and hyperostoses were seldom seen. His experience was that operative removal of exostoses was rarely called for.

Mr. A. CHEATLE said he had examined post mortem the temporal bones of about 900 hospital patients, and had found the usual triple deep sessile exostoses in only one case, and general thickening of the tympanic plate or hyperostosis in another.

The PRESIDENT agreed with Dr. Pritchard's remarks as to frequency. Such cases were more frequent in the South, and more so among individuals who had lived in hot climates. Fifteen or sixteen years ago he showed drawings of a dozen cases, and such growths seemed so comparatively common that he ceased to have drawings made of them. None of them had been operated upon, nor had any urgent symptoms. A causative factor seemed to be the pouring of water into the ears, especially carelessly sponging while holding the head on one side. Some individuals were not satisfied with the

ordinary bath, but liked to hold their heads under the water. Operative treatment was rarely necessary except for patients who lived in regions far removed from expert surgical aid.

Dr. MCKENZIE reminded the Section that at the Belfast meeting of the British Medical Association, Dr. Jackson, of Plymouth, had read a paper¹ on the subject, showing that the presence of such tumours was associated with residence at or near the sea-coast. Probably salt water had some effect in their production. In the present week he had seen a case of hyperostosis in private. He could not answer the patient's question as to whether the growth would be rapid or slow, but he believed it would be slow.

Dr. WESTMACOTT said he remembered only three cases in the last twelve years in private work. The condition had been aggravated, and the patient had sought relief on account of the accumulation of wax, which interfered with hearing. One case had remained unaltered for eight years, though he had only a very small opening. He refused to operate in every case, as he did not think it necessary.

Mr. TOD, in reply, said he showed the patient's case because he had never before seen an exostosis occurring in so young a subject. He was sure that there was no perforation of the tympanic membrane, and the hearing was normal. He agreed that the growth might, in this case, be removed through the external auditory canal, but he, personally, had always operated by making a post-auricular incision and turning the auricle forward. His experience was based on six cases—three in hospital practice and three in private. In each of the hospital cases the exostosis was single and pedunculated, and arising from the posterior wall of the auditory canal. In the private cases there was complete obstruction of the external meatus, the growths being sessile and multiple. He agreed that the operation was only justifiable in extreme cases, such as when there was complete deafness of one side with exostoses almost blocking up the other ear. In two cases the patients themselves insisted on operation owing to the pain set up in the ear by the exostoses impinging against one another, and causing superficial ulceration and inflammation. The cases in which the exostosis was single and pedunculated were easy; those in which the growths were multiple were very difficult. In the latter group of cases the growths bordered on the membrane, and in every case the tympanic membrane was injured by operation. The results, however, were uniformly good, the tympanic membrane healing, and the hearing power being restored to normal. He agreed with the President that it was not so uncommon to see narrowing of the meatus, owing to the presence of hyperostosis, especially in patients getting on in years, often with a gouty history, and in those who suffered from chronic middle-ear progressive deafness.

¹ *Brit. Med. Journ.*, 1909, ii, p. 1137.

A Case in which the Clinical Symptoms simulated a Cerebellar Abscess. Brain explored on two occasions. No Abscess discovered. Recovery.

By HUNTER TOD, F.R.C.S.

THE patient, a girl aged 23, had a double mastoid operation performed in March, 1909. She was extremely deaf before the operation, and equally so afterwards, so that she could only hear when shouted at. The right side healed well; on the left side there was some contraction of the auditory canal. During the last few months there had been attacks of giddiness, with occasional vomiting, the latter becoming more frequent lately, and there had been almost constant headache, referred chiefly to the left side, although occasionally to the frontal region. There was slight and intermittent nystagmus towards the left side. Owing to the continual complaints of headache and the increasingly ill-appearance of the patient, she was again taken into the hospital. I thought the symptoms might be due to retention of pus, as a result of stenosis of the auditory canal. There was facial paralysis, which had existed for some long period.

On March 2, 1910, an incision was made along the post-auricular scar, and the mastoid cavity exposed. There was no appearance of disease, the cavity being lined with skin, excepting over the promontory, the surface of which seemed to be rough. The inner wall of the tympanic cavity and this region were carefully examined, and no fistula could be discovered either through the promontory itself or in the external semicircular canal. Surgical procedure was limited to dilating the auditory canal and to making a posterior meatal-flap, the wound cavity being left open and lightly packed with gauze. Twenty-four hours after the operation there was pyrexia of 102° F., accompanied by all the signs of cerebral irritation. There was marked vomiting, increased nystagmus to the left, and increased reflexes. The intellect, however, was clear. This was succeeded rapidly by symptoms of intracranial pressure, the pulse becoming reduced to 60 or less, and the temperature to 97.6° F. The patient became drowsy and lay on the affected side with the knees drawn up, and refused to answer questions.

Diagnosis of cerebellar abscess was made. Thirty-six hours after the previous operation the cerebellum was explored by the route

anterior to the lateral sinus. On incising the dura mater there was obvious increase of cerebrospinal fluid. The brain substance was incised in various directions with a negative result. The temporo-sphenoidal lobe was also explored with a similar result. The wound was then packed with gauze. Twelve hours later the patient seemed to be less drowsy and more sensible, but answered questions very slowly. The nystagmus had now almost diminished, and there was no further complaint of giddiness. The left arm was more flaccid, and the left hand-grip weaker than on the right side. Romberg's rotatory phenomenon was present in the left arm and hand. There was blurring of the left optic disk, but no marked optic neuritis.

As the symptoms of intracranial pressure continued, the cerebellum and temporo-sphenoidal lobe were again explored on March 7, with a negative result. The wound kept clean and there was no hernia cerebri. The patient remained in the same state for two or three days, then gradually improved. Suddenly, a week after the last exploration, she said she felt something go "pop" in the left side of the head, and after this she could hear very much better. From this time onwards she progressed favourably, and regained her normal movements in the left hand and arm. On March 30, although apparently well, she said she was not able to read, although she was able to crochet. Previous to the operation she could read perfectly. The swelling of the optic disk gradually disappeared and the fundi became normal.

The case is of interest in that it simulated so markedly a cerebellar abscess. A suggested explanation is that as a result of opening up the mastoid the labyrinth became infected, and as a sequela to this there was a localized accumulation of cerebrospinal fluid in the posterior intercranial fossa, which was drained by the operative procedures. The second point requiring explanation is the distinct improvement of hearing occurring immediately after the patient's subjective sensation of something going "pop" in her head.

Dr. DAN MCKENZIE said that some six months previously he had seen a case in which symptoms simulating brain-abscess had come on a few days after the radical mastoid operation. The patient became dull and apathetic, answering questions slowly, the pulse and temperature sank to below normal, and the pupils reacted very sluggishly. Marked nystagmus to the affected side seemed to confirm the fears of an intracranial disaster. But, as it was not clear whether the cerebrum or the cerebellum was affected, operation was postponed for twelve hours. At the end of that time, however, the patient

had decidedly improved; the intellectual dullness passed away, the pulse and temperature rose to normal, and he made a good recovery. The nystagmus persisted for several days. This and other cases had taught him that spontaneous nystagmus of itself should not be regarded as a reliable guide to the situation, character, or severity of a lesion.

Specimen of Cholesteatoma or Keratosis Obturans of the External Auditory Canal.

By HUNTER TOD, F.R.C.S.

THE patient, a man aged 48, complained of deafness and earache, which had been made worse by syringing. Examination showed a large white mass filling up depths of the auditory canal. There was no history of otorrhœa. Instillation of hydrogen peroxide lotion, followed by prolonged syringing, failed to remove the mass. Further treatment was carried out at home, by the patient's medical man, but with negative result. He tried in turn solutions of bicarbonate of soda and glycerine, carbolic oil, and rectified spirits, together with repeated syringings. Two weeks later the patient returned to me, and with great difficulty, by means of repeated syringing and the use of instruments, I removed the mass shown. It was removed in three portions, and, when fresh, consisted of an outer glistening layer, and when first placed in water it expanded, and its structure seemed to consist of concentric layers of tissue closely pressed together. Further examination of the ear now showed the inner portion of the auditory canal to be larger than normal, and lined with desquamating epithelium, as was also the depressed surface of the tympanic membrane. I was surprised to find the tympanic membrane quite intact.

The patient now hears normally, but has to be seen at intervals of six months, as there is still a tendency for re-accumulation of masses of desquamating epithelium.

Two Cases of Cholesteatoma of Unusual Size extending into the Posterior Fossa and causing Obliteration of the Sigmoid Sinus.

By GEORGE WILKINSON, F.R.C.S.

CASE I.

J. W., AGED 49, a labourer, was admitted to the Sheffield Royal Hospital from the out-patient department on November 10, 1909. He was extremely deaf, and had to be communicated with by writing. There was offensive purulent discharge from both ears, and complete flaccid paralysis of the right side of the face. He complained of great pain in the right occipital region and vertex, on the right side of the middle line.

The history was difficult to obtain owing to the patient's deafness. He dated the beginning of his ear troubles to a fall on his head sixteen years previously. He was unconscious for about an hour after the accident. He thinks there was no discharge of blood from the ears. He believes he was slightly deaf in the right ear before the accident. About three months later he noticed that he was more deaf on the right side, and he began to have a discharge from the right ear. He does not know when the discharge from the left ear began. The face became "drawn" thirteen years ago, when he had an attack of pain in the right ear. He has had attacks of pain from time to time, but the pain has been much worse during the last two months.

November 10, 1909. Right ear: Profuse offensive, thin, blood-stained discharge from the ear. The depth of the meatus is filled with granulations; a probe passed into the meatus enters a cavity beyond, causing pus to well up between the granulations. The right ear appears to be absolutely deaf. A tuning fork (C_1) struck loudly is heard for a few seconds when placed on the mastoid, probably by conduction to the opposite side. Left ear: Profuse purulent discharge. The inner tympanic wall can be seen through a large perforation in the membrana tympani. He can occasionally catch a word if one shouts loudly close to the ear. Bone conduction (C_1) is lengthened five seconds. Vestibular reaction: Rotatory—right ear absent, left ear present (? twenty seconds after ten rotations in twenty seconds); Caloric—right ear absent, left

ear well marked; appeared after 25 seconds' irrigation and lasted 115 seconds after one-minute irrigations with water at 70° F. Gait quite steady. No Romberg symptom. Jumps backwards with eyes closed without staggering afterwards.

Operation (November 11) on the right ear. Usual mastoid incision. Mastoid cortex very dense, $\frac{3}{8}$ in. thick. As soon as the cortex was penetrated by the chisel, very offensive pus welled up under pressure. Removal of the cortex exposed a large cavity filled with decomposing cholesteatoma, partly laminated, but mostly pultaceous, mixed with pus and small sequestra. The posterior wall of the osseous meatus was chiselled away, exposing the tympanic cavity. The inner tympanic wall was much eroded, and all the bone posterior to the antrum had been absorbed, leaving a large opening into the sinus groove and posterior fossa. The facial canal could not be identified. An open vertical groove on the inner wall of the aditus was probably the remains of the superior semicircular canal. The vestibule, the facial canal, and the external semicircular canal had apparently entirely disappeared. Posteriorly the dura mater and sigmoid sinus was pushed backwards by a mass of cholesteatoma which had invaded the posterior fossa. The resulting cavity extended in a downward, inward, and forward direction towards the base of the skull, in which direction a probe could be passed into the depth of the wound 7.5 cm. from the surface of the mastoid. The dura mater was much thickened, and the sigmoid sinus appeared to be entirely obliterated down to the jugular foramen. A large meatal flap was made, and the mastoid wound closed. The cavity was packed with gauze through the meatal wound. The large cavity, with its rigid wall of bone and thickened dura mater, filled up very slowly. Injections of Carl Beck's bismuth wax were made into the post-aural cavity on January 11 and 25 and March 1.

He has been free from pain since about a week after the operation. The post-aural cavity is nearly obliterated, there being only a small amount of discharge daily. The hearing of the left ear has much improved under regular cleansing. It is possible to make him understand anything that is said to him by shouting.

CASE II.

C. R., aged 18, was admitted to the Sheffield Royal Hospital, December 14, 1909. Profuse discharge of somewhat offensive pus from the left ear, some headache, and complete left-sided facial paralysis.

Discharge from the ear began after measles in early childhood. Thirteen years ago she had a mastoid operation performed (not by reporter). A certain amount of discharge has continued ever since. The face began to be "drawn" eight weeks before admission, and a few days later she had some pain in the ear. The discharge had ceased, but began again a few days later, and the pain was relieved, though she had some headache.

Examination of the left ear shows the meatus to be narrowed to a mere sinus half an inch within the orifice. In the scar of the old mastoid wound is a small sinus discharging a little pus. There is complete paralysis of the left side of the face, but none of the soft palate or uvula.

Operation, December 15: Mastoid exposed through the old scar. The sinus led into a large cavity, the expanded antrum and tympanum in one. There was a free opening posteriorly into the sigmoid sinus groove. The whole cavity was filled with laminated, firm cholesteatoma, and a certain amount of pus. The cavity in the mastoid was fully exposed by removal of further portions of the mastoid cortex, and the groove for the sigmoid sinus was freely opened up. The inner tympanic wall was covered with firm, organized granulation tissue, so that the details of its condition could not be made out. The deeper meatus was partially lined with skin, the desquamating epithelium from which had no doubt given rise to the cholesteatoma, its escape from the ear being cut off by the contraction of the meatus. The point at which the facial nerve was being compressed could not be made out. No opening in the facial canal could be detected.

The sigmoid sinus groove was entirely filled with cholesteatoma, and the sinus itself obliterated. The cholesteatoma was removed by lifting it out of its bed gently with a blunt spoon. The last portion to be removed filled the lowest portion of the groove in the neighbourhood of the jugular foramen. As it was being lifted out a great gush of venous blood welled up, showing unmistakably that the sinus had been opened. The hæmorrhage was immediately controlled by plugging. All remnants of skin were curetted out from the tympanum and deep part of the meatus, and the mastoid wound left open and packed, with the object of obtaining obliteration of the whole cavity. Smart hæmorrhage occurred from the sinus when the packing was removed two days later, and was again arrested by packing.

When the cavity was granulating, hearing and vestibular tests were carried out, with the following results: Left ear—mastoid, Co $\frac{7}{60}$, C₁ $\frac{7}{30}$,

$C_2 \frac{30}{30}$, $C_3 \frac{12}{17}$, $C_4 \frac{3}{8}$; aerial conduction, $Co 0$, $C_1 0$, $C_2 \frac{12}{15}$, $C_3 \frac{7}{30}$, $C_4 \frac{4}{20}$. Right ear—hearing quite normal. Rotatory: Left ear—slight nystagmus for a few seconds; right ear—nystagmus for twenty seconds after seven rotations in twenty seconds. Caloric: Left ear—no nystagmus; right ear—nystagmus induced after fifteen seconds' irrigation; lasted sixty seconds after one-minute irrigations with water at 70° F. Romberg symptoms—slight swaying; sways after jumping backwards with eyes closed.

Second operation, February 2, 1910: No improvement having taken place in the facial paralysis, it was decided to do Alt's operation. The facial nerve was exposed at the stylo-mastoid foramen, and by chiselling away the overlying bone was traced upwards as far as the inner tympanic wall, where it merged in a mass of scar-tissue. Erosion of the inner tympanic wall had taken place at this point, destroying the nerve and probably also the vestibule. The scar-tissue was scraped away, and the exposed nerve covered with a fragment of gold leaf, over which a piece of rubber tissue was laid. It was hoped that new nerve fibrils from the proximal end of the nerve might find their way to the exposed distal end. Up to the present no improvement in the facial paralysis has taken place. The mastoid wound has healed, and the auditory meatus is closed $\frac{1}{2}$ in. from its orifice.

Malignant Growth of the Right Temporal Bone, with Extension through the External Meatus, resembling an Aural Polypus.

By P. WATSON WILLIAMS, M.D.

W. W., MALE, aged 57, was referred to my aural clinic at the Bristol Royal Infirmary by Dr. Rattray, of Frome, with a polypoid growth protruding from the right external auditory meatus. Its surface was black from congealed blood, an attempt having been made to snare the polypus by Dr. Rattray.

History: The patient first noticed increasing deafness in May, 1909, and, thinking it was due to wax, tried olive-oil drops. No noticeable development occurred till August, when he began to suffer from pains in the head and giddiness, which prevented him working. In September he discovered a small, smooth swelling in the head, in October a lump over the left fifth rib, and in November a swelling on the right buttock.

These swellings have increased, but have remained painless. About Christmas-time he first noticed something coming out of his right ear, which bled when he tried to scratch it away, and facial paralysis also began to develop then. On admission it was complete, with absolute deafness in the right ear. There was no history of syphilis.

Portions of the growth protruding from the external meatus were removed and submitted to Professor Walker Hall, pathologist to the Royal Infirmary, but he was unable to report definitely as to the nature of the growth. Small fragments removed on February 28, 1910, were found to be covered with epithelium, showing no malignant change, with subdermal tissue of collections of round-cells, difficult to differentiate from sarcomatous, but "if all the polypi were removed it is most probably innocent." Later a larger mass removed was pronounced undoubtedly malignant. As the pathological report is appended in detail, further reference to these points is unnecessary here. He was put on iodide of potassium, 40 gr. daily.

March 9: Mental condition seemed little affected, but he was weaker and losing weight. An area of dullness, with moist sounds and increased vocal resonance, developed over the base of the left lung.

Ocular conditions: Right eye—some central cataract and old diffuse chorio-retinitis; left—disk pallid, no neuritis.

A curvilinear incision through the skin was made over the right mastoid down to the bone, which was very thin, and the vascular friable growth exposed; but as it was obviously impossible to remove it, the wound was immediately closed. He became progressively weaker, and died on March 17.

The following specimens were shown:—

(a) Kidney, showing subscapular "mixed nephroma." The tumour was partly encapsulated and alveolar in type. At one pole it infiltrated the renal cortex, and its extension into the venous channels was shown. Microscopical sections showed: (1) Columns and masses of large, clear, granular cells and their relations to blood-vessels; (2) hæmorrhages; (3) cell degeneration.

(b) Rib, showing invasion and replacement of the bony and medullary tissues by secondary growths. Microscopical specimens showed: (1) Cells sarcomatous in type and arrangement; (2) cells large, clear, arranged in masses; (3) tubules, lined by darker, smaller cells; (4) hæmorrhages.

(c) Right parietal bone, showing involvement of both tables by secondary nephroma growths.

(d) Right temporal bone opened to show the extent of invasion and replacement by secondary nephroma growths.

(e) Microscopical specimens of skull showing (1) masses of large, clear cells, arranged in alveoli or upon papillomatous projections in close relation to blood-vessels; (2) giant cells, containing numerous nuclei; (3) hæmorrhages; (4) cell-rich stroma.

PATHOLOGICAL REPORT BY PROFESSOR WALKER HALL.

At the autopsy the chief feature was the presence of growths in the left kidney, the ribs, lungs, and skull. Except for slight bilateral pleuritic exudates and a fatty heart, with early aortic atheroma, the other findings were not noteworthy. The renal growth was well encapsuled, except at one area, where it was invading the renal cortex and extending along the renal veins. The growths in the ribs involved the third on the right side and the fourth and fifth on the left side; they bulged externally and internally, and replaced the bony and medullary tissues entirely. Of those in the skull, one was situated near the central border of the right parietal bone; it was about 2 in. in diameter, and had destroyed both the inner and outer tables. The other tumour extended from the outer margin of the right petrous bone inwards along its whole length to the right lobe of the cerebellum. A probe inserted into the right auditory meatus passed easily through the growth to the cerebellar area. The meninges were locally involved. No cerebral metastases were found. All the growths were soft and hæmorrhagic, and to the naked eye they presented similar appearances.

Microscopically each tumour showed slight local modifications, but the main features were identical in each instance. These features may be summarized as follows:—

(1) Columns and masses of cells having definite relations to neighbouring capillaries. The cells are suprarenal in type, clear, cubical, and exhibit proliferative changes. In some parts there is fusion to form large masses of cytoplasm containing numerous nuclei. Fat and glycogen are present.

(2) Tubules, whose cells are smaller and less transparent.

(3) A stroma rich in blood-vessels and in places surrounding the cells so as to give an alveolar appearance.

(4) Hæmorrhages, fatty metamorphoses and calcareous depositions.

Taking the renal nodule as the most characteristic, sections from the ribs show a comparatively large percentage of tubules and a tendency to

sarcomatous changes not elsewhere exhibited. Those from the skull present tubules and masses of cell in equal proportions, and show many fused cells and areas in which the cells are elongated and arranged in rows upon a central core of blood-rich stroma.

In the main the appearances are those of a hypernephroma, but there are some renal elements also; and the condition must be regarded as due to the growth of bony metastases from a mixed nephroma.

Specimen of Fracture of the Temporal Bone.

By G. J. JENKINS, F.R.C.S.

THE fracture involves the roof of the Eustachian tube and tympanic cavity, and squamous temporal. Externally the fracture extends to the under surface of squamozygomatic element immediately behind the postglenoid tubule along the area of articulation with the tympanic plate. The middle-ear tract is filled with blood-clot. The tympanic membrane is unruptured, and there was no blood in the meatus. This specimen affords an explanation of an uncommon clinical condition, bleeding into the middle-ear tract without rupture of the tympanic membrane, in fracture of the base of the skull.

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ROYAL SOCIETY OF MEDICINE

VOLUME THE THIRD

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

SURGICAL SECTION



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CONTENTS.

July 13, 1909.

	PAGE
The Treatment of Inoperable Sarcoma by Bacterial Toxins (the Mixed Toxins of the <i>Streptococcus erysipelas</i> and the <i>Bacillus prodigiosus</i>). By WILLIAM B. COLEY, M.D. (New York)...	1

October 12, 1909.

Prognosis in certain Inflammatory Diseases of the Lungs and Pleura commonly treated Surgically (Presidential Address). By RICKMAN J. GODLEE, M.S....	49
--	----

December 14, 1909.

A Discussion on "The Diagnosis and Treatment of Duodenal Ulcer."	
Introductory Address by B. G. A. MOYNIHAN, M.S. ...	69
Sir LAUDER BRUNTON, F.R.S. (p. 87)—Dr. W. HALE WHITE (p. 89)— Mr. H. WATERHOUSE (p. 91)—Mr. FREDERIC EVE (p. 93).	

January 11, 1910.

Discussion on "The Diagnosis and Treatment of Duodenal Ulcer" (<i>continued</i>).	
Dr. ROBERT HUTCHISON (p. 97)—Dr. A. F. HERTZ (p. 100)—Mr. HERBERT PATERSON (p. 107)—Dr. NATHAN RAW (p. 111)—Dr. CRAVEN MOORE (p. 111)— —Mr. C. P. CHILDE (p. 113)—Mr. MOYNIHAN (p. 114).	

February 8, 1910.

The Radical Operation for Cancer of the Pylorus, with especial reference to the Advantages of the Two-stage Operation and to the Question of the Removal of the Associated Lymphatics. By ERNEST W. HEY GROVES, M.S. ...	117
--	-----

March 8, 1910.

	PAGE
Rupture of the Tunica Vaginalis in Hydroceles. By SOMERVILLE HASTINGS, M.S.	165
The Clinical Significance of Gastric Hypersecretion and its Connexion with Latent Disease of the Appendix. By W. SOLTAU FENWICK, M.D.	177
Appendicular Gastralgia, or the Appendix as a Cause of Gastric Symptoms. By HERBERT J. PATERSON, F.R.C.S.	187

June 7, 1910.

The Present Position and Treatment of Syphilis. A Discussion, opened by J. ERNEST LANE, F.R.C.S.	209
Dr. ALEXANDER FLEMING (p. 219)—Major H. C. FRENCH (p. 222)—Capt. L. W. HARRISON (p. 226)—Mr. J. E. R. McDONAGH (p. 228).	

June 14, 1910.

Discussion on the Present Position and Treatment of Syphilis (<i>continued</i>)	233
Mr. C. F. MARSHALL (p. 233)—Dr. GEORGE PERNET (p. 234)—Mr. H. W. BAYLY (p. 238)—Mr. JONATHAN HUTCHINSON (p. 242)—Mr. ERNEST LANE (p. 246).	

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Surgical Section.

July 13, 1909.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

The Treatment of Inoperable Sarcoma by Bacterial Toxins (the Mixed Toxins of the *Streptococcus erysipelas* and the *Bacillus prodigiosus*).

By WILLIAM B. COLEY, M.D. (New York.)¹

THE subject upon which I have been asked to address you is one upon which I have been working constantly the last seventeen years, and one which has grown more interesting to me with each succeeding year. While the results have not been as satisfactory as one who is seeking perfection could wish, they have been sufficiently real and tangible, I think, to be entitled to more careful consideration than they have yet received. Furthermore, they may have an important bearing upon the whole cancer problem, since, if by the administration of certain bacterial toxins we can cause the degeneration, death, and absorption of living tumour cells of one variety of cancer—sarcoma—it is not unreasonable to suppose that by the use of some other forms of bacterial toxins we may succeed in destroying or inhibiting the growth of the other and more common variety—carcinoma.

At the outset I wish, at the risk of tiring the few of you who may have read and remember some of my former papers upon this subject, to give in the briefest possible words an outline of the early history of the method of treatment, and the several stages of its development, for the benefit of the many who have never heard of the mixed toxins of

¹ Professor of Clinical Surgery at Cornell University.

erysipelas and *Bacillus prodigiosus*, or whose knowledge of the same is of the most general sort. Such early history of the method is essential for the proper consideration and understanding of its present status.

First, I wish to emphasize the point that the method rests upon a solid foundation of accepted and indisputable clinical facts—namely, that in a considerable number of cases of inoperable cancer of all varieties, and especially sarcoma, such tumours have been known entirely to disappear under attacks of accidental erysipelas, and patients have remained well for many years thereafter.

For those who refuse to accept clinical results unless confirmed by laboratory experiments, these latter tests have now been supplied, since during the last two years Dr. Martha Tracy and Dr. S. P. Beebe, of the Huntington Cancer Research Fund, have shown that large multiple sarcomas in dogs rapidly disappear under both local and systemic injections with the mixed toxins of erysipelas and *Bacillus prodigiosus*.

My attention was first called to the curative effect of accidental erysipelas in inoperable sarcoma by a certain case observed in 1891. In my studies of sarcoma at that time I made a careful analysis of all the cases of sarcoma (90 in number) operated upon at the New York Hospital during the preceding fifteen years. Among these cases was a small round-celled sarcoma of the neck, four times recurrent. At the fifth operation, in 1884, Dr. Bull found the tumour to involve the deep structures so extensively that it was impossible to remove it, and he gave up the attempt. The case was regarded as absolutely hopeless, when, shortly after the operation, the man developed a very severe attack of accidental erysipelas in the face and neck, followed two weeks later by a second attack. Within a few days after the beginning of the first attack the tumour began to soften and decrease rapidly in size. The history stated that when the patient left the hospital his tumours had entirely disappeared. There was no after-record of the case, but I made an effort to trace the patient, and finally found him alive and well, with no evidence of any local or general recurrence in the spring of 1891, seven years later. He was examined both by Dr. Bull and myself. (Fig. 1.)

At this time I had not read of Fehleisen's experiments in Germany in inoculating patients with inoperable malignant tumours with the streptococcus of erysipelas; but I was so strongly impressed with the case I have related that I determined to try inoculations in the first suitable case. In a very short time—May 2, 1891—I made my first

inoculation in a case of recurrent spindle-celled sarcoma of the tonsil and neck, kindly referred to me by Dr. William T. Bull. The patient was an Italian, aged 35, first operated upon for sarcoma of the tonsil and neck by Professor Durante, of Rome, in 1890, and again by Dr. William T. Bull, at the New York Hospital, in April, 1901. The tumour was found much too extensive for removal, but a portion was excised for microscopical examination, which proved it to be a spindle-celled sarcoma. At the time of my first inoculation there was a tumour



FIG. 1.

Recurrent round-celled sarcoma. Spontaneous recovery following accidental erysipelas. Photograph taken seven years after the cure.

of the right tonsil, nearly as large as an egg and almost completely blocking the pharynx; there was also a large metastatic tumour in the right cervical region. The patient could take no solid and little liquid food, and was much emaciated and cachectic. The details of this case were published in my first paper, "A Contribution to the Knowledge of Sarcoma."¹ The history of the case in brief is as follows: I worked

¹ *Ann. of Surg.* St. Louis, 1891, xiv, p. 199; and also *Amer. Journ. Med. Sci.*, Philad., 1893, cv, p. 487.

continuously from May to October, 1891, to produce an attack of erysipelas, without success. Cultures from four different laboratories were used and various methods of inoculation employed. Finally, in October, 1891, with 5 decigrams of a bouillon culture of streptococcus of erysipelas, just brought me from Koch's laboratory in Germany by Dr. Frank Ferguson, the pathologist of the New York Hospital, a most severe attack of erysipelas developed, nearly causing the death of the patient. Within an hour after the injection a severe chill occurred, followed by a temperature of 105° F. After an interval of twelve hours a typical attack of erysipelas developed, starting at the point of injection and extending over the neck and face. It ran its usual course. The tumour of the neck began to break down on the second day, and a discharge of broken-down tumour tissue continued until the end of the attack. At the end of two weeks the neck tumour had disappeared and the tonsil tumour had decreased in size. The patient remained well for eight years, and then died in Italy of a local recurrence.

During the next two years, through the kindness of Dr. Bull, I had an opportunity of trying the inoculations upon a number of chronic and incurable cases of malignant disease at the New York Hospital, in a special building erected for the purpose by Mr. Archer M. Huntington. It is necessary to refer to these earlier investigations with the inoculations of the living cultures, inasmuch as they form the basis of all the later work with the toxins.

Of the first ten cases treated by the living cultures (local and systemic injections of bouillon cultures of the streptococcus of erysipelas) six were sarcoma and four carcinoma, all inoperable and far advanced. In seven of the ten cases I could not produce erysipelas after many attempts extending over many weeks. In all these cases of failure to produce erysipelas, however, I noticed slight temporary improvement in the tumours as shown by decrease in size, increase in mobility, and diminution of vascularity. In only three cases of sarcoma was it possible to produce an active attack of erysipelas; one of these I have already related in brief. The second was a large inoperable sarcoma of the back (mixed-celled) with extensive metastases in the right groin of a man, aged 46. The tumour of the groin was partially removed, but recurred quickly. Specimens from the primary tumour of the back were excised and pronounced mixed-celled sarcoma by Dr. Frank Ferguson, pathologist to the New York Hospital, and the diagnosis was confirmed by microscopical examinations made by a number of other pathologists. I first

inoculated him on April 21, 1892. Injections with the living bouillon cultures of the streptococcus of erysipelas obtained from the laboratory of the College of Physicians and Surgeons were repeated daily for three weeks without causing an attack. In the fourth week an injection of 20 mm. of a fresh culture obtained from the Johns Hopkins Hospital Laboratory was given without effect. A second injection, two days later, was followed by a severe chill and a temperature of 105.5° F. Twenty-four hours later a typical area of erysipelas developed. To quote from my paper (loc. cit. 1893): "From the beginning of the attack the change that took place in the tumour was nothing short of marvellous. It lost its lustre and colour and had sunk visibly in size within twenty-four hours. Several sinuses formed the second day and discharged necrosed tumour-tissue. A few days later the tumour of the groin, which was about the size of a goose egg and very hard when the inoculations were begun, broke down and discharged a large amount of tumour-tissue. Three weeks from the date of the attack of erysipelas both tumours had entirely disappeared. July 1, two months later, there was a small local recurrence in the back. Two weeks later six small nodules had developed. Inoculations were resumed, but no attack of erysipelas could be produced until November 14, 1892, although constant attempts were made. By October 1 the tumour had been 2½ in. by 4½ in., and by November 14 5 in. by 3½ in., and there was a marked recurrence in the groin. On November 14, after an injection of 22 mm. of a culture previously used without effect, a moderately severe attack of erysipelas developed, during the course of which the tumours both in the back and groin disappeared. In two weeks there was evidence of recurrence. Curiously, during the next three months, three further attacks of erysipelas developed spontaneously; they were milder in character, and the effect upon the tumours was less pronounced and less lasting." Finally, early in 1893, under repeated injections of the mixed toxins of erysipelas and *Bacillus prodigiosus*, the tumour disappeared and the patient remained well for three and a quarter years, when he died of a malignant tumour of the abdomen, probably metastatic.

The third case of sarcoma in which I produced an attack of erysipelas was a twice-recurrent sarcoma of the breast in a woman aged 38, referred to me by Dr. Bull. An inoculation given June 2, 1892, resulted in a moderately severe attack of erysipelas. All of the three tumours present decreased considerably in size; one almost disappeared. A second attack of erysipelas was produced on June 30; the tumours showed some further improvement, but less than from the first attack. The check in

growth proved but temporary, and the tumours soon began to increase in size. In spite of further inoculations the patient grew worse and died within six months.

In none of the cases of carcinoma did I succeed in producing erysipelas. Although up to this time I had had no death, the difficulties in the way of successful inoculations were very great. Shortly afterward I had two deaths, both in far-advanced cases, and the patients had been duly warned of the risks of inoculation. These experiments with the living cultures, while absolutely confirming the curative influence of accidental erysipelas, as shown by clinical observation, also emphasized the practical difficulties which had been sufficient to cause Fehleisen to abandon his attempts. I had learned from the same source:—

- (1) That it is extremely difficult to produce erysipelas at will;
- (2) That the risks of inoculation, when successful, were considerable;
- (3) And, most important, I had been impressed with the fact that repeated injections of the living bouillon cultures of the streptococcus of erysipelas had an inhibitory action upon the growth of the tumours which, while only temporary, was nevertheless distinct.

This fact led me to believe that a portion at least of the curative action of the erysipelas lay in the toxic products of the erysipelas, which might possibly be utilized without producing an actual attack of erysipelas.

In the latter part of 1892 I made my first experiments with the toxins of erysipelas. I began with bouillon cultures of the streptococcus of erysipelas, sterilized by just sufficient heat (58° C.) to destroy the germs, and also by filtering the unheated cultures through a porcelain filter. These toxins were prepared for me by Dr. Alexander Lambert. Four cases of inoperable sarcoma were treated by this preparation with constitutional reactions very similar to those obtained from the living cultures. The temperature would rise to 103.5° F., but would always fall to normal on the following day. There was some inhibitory action on the tumours, but this was temporary. I obtained cultures from fatal cases of erysipelas, in order to get the highest degree of virulence.

At this time, Roger's experiments with the prodigious cultures showed that if the *Bacillus prodigiosus* were grown together with the streptococcus of erysipelas the virulence of the latter was materially increased. Roger had never used the *Bacillus prodigiosus* alone or with the streptococcus of erysipelas on the human being, and had never, as far as I know, suggested it as a therapeutic agent.

In order to intensify the virulence of the erysipelas, I decided to use

the combined toxins of erysipelas and *Bacillus prodigiosus*, growing the two organisms together and sterilizing them with heat. The first preparation was made for me by Dr. B. H. Buxton, then Fellow of Bacteriology of the Loomis Laboratory, and now for seven years Professor of Experimental Pathology of Cornell University. The erysipelas culture was grown alone in bouillon for ten days, then the *Bacillus prodigiosus* added, the two grown together for ten days, and then sterilized by heating to 58° C., and kept sterile by the addition of a little thymol. This is the preparation that was used with little change until three years ago, when Dr. Martha Tracy, working with Dr. Buxton, suggested an important modification. Her subsequent experiments proved the truth of the opinion that I had already expressed some time before, based upon clinical observations alone—that the *Bacillus prodigiosus* had in itself a curative effect upon tumours, independent of any action it might have in intensifying the virulence of the erysipelas.

Tracy's first experiments with the *Bacillus prodigiosus* alone confirmed the investigations of Vaughan, of Ann Arbor—that the *Bacillus prodigiosus* toxins were the most powerful known. Further experiments with sarcoma in dogs showed that sarcoma would disappear under the injections with the prodigiosus alone, without any erysipelas, although not quite so rapidly as when the combined toxins were used. Utilizing these newly proven facts, Dr. Tracy proceeded to grow the two organisms separately and, by adding a certain definite quantity of the sterilized prodigiosus bouillon to each ounce of the streptococcus broth, was able to secure what had never been possible before—namely, a definite standardization of dosage. This enabled us to overcome the greatest difficulty we had had to contend with all along.

In the old way of growing the two organisms together, there had always been a varying amount of prodigiosus toxins in the solution, due to an exceedingly variable rate of growth. I had previously noted clinically that the highly coloured solution of the toxins, showing a large amount of prodigiosus present, were more powerful and, likewise, the curative effect was greater. The first preparations which Dr. Tracy made in the way described had so large an amount of prodigiosus that very severe reactions were obtained by minute doses, and in one case, in the hands of another physician, death resulted within a few hours after an injection of $\frac{1}{4}$ mm. made into a very vascular tumour in the mediastinal region. After this the addition of the prodigiosus to the toxins was immediately reduced to one half, and this amount has been continued up to the present time.

The process of preparation is as follows:—

To Prepare the Streptococcus Broth.—Soak 1lb. minced beef overnight in 1,000 cc. of cold water. Then boil for one hour and filter through coarse cotton cloth of any sort. Add of peptone (Witte's) 10 gm.; of NaCl (sodium chloride) 5 gm. Test the reaction to litmus and render slightly alkaline by addition of a sufficient quantity of 10 per cent. NaOH (sodium hydroxide) solution. Boil for one hour. Filter through filter paper. Distribute into small flasks, 25 to 50 cc. in each flask. Sterilize by boiling for one half-hour on three successive days. Sow each flask with a few cc. of a broth culture of streptococcus.¹ Allow to grow in the incubator for three weeks.

To Prepare the Prodigiosus Suspension.—Spread an ordinary 2 per cent. agar medium to a depth of about 1 cm. at the bottom of a large "Roux" or "antitoxin" culture flask. Sterilize as usual by boiling for one half hour on three successive days. Over the surface of the agar, with the usual precautions against contamination, pour a two-day-old broth culture of *Bacillus prodigiosus*.² Manipulate the flask so that the entire agar surface has been touched by the broth, and drain off the surplus fluid. Allow the prodigiosus to grow at room temperature in daylight, but protected from the direct sunlight, for ten days. Scrape off the thick red growth with glass rods and rub up with a pestle and mortar to a smooth, rather thick suspension, using physiological salt solution as diluent. Bottle and sterilize in the bottle, by heat, at 75° C., for one hour. This suspension can be diluted further at any time. The amount of diluent needed is ascertained by determining the weight of nitrogen per cubic centimetre of suspension (Kjeldahl's method). This multiplied by 6.25 gives the weight of proteid present, and this should be 12.5 mgm. per cubic centimetre of the suspension to be used for the mixture.

To Prepare the Mixture.—Take of streptococcus broth culture, three weeks' growth, 100 cc.; of prodigiosus suspension (containing 12.5 mg. of proteid per cubic centimetre or 375 mg.³ of proteid in all) 30 cc.; of glycerine, 20 cc.

¹ The streptococcus used during the past two years in the laboratory of the Huntington Cancer Research Fund was isolated from a fatal case of septicæmia. It is doubtful whether an organism from an actual case of erysipelas would give any better results. The stock culture of streptococcus has been maintained more satisfactorily in broth than on agar. No attempt has been made in recent work to keep up the virulence by passing through animals.

² The stock culture of prodigiosus is kept upon agar slants, a tube of broth being inoculated from the agar two days before it is needed for the large flasks.

³ It was found desirable to reduce the amount of prodigiosus proteid to one-half of the amount shown in the formula given in my paper published in the *Medical Record*, New York, 1907, lxxi, p. 436.

Each cubic centimetre of the mixture then contains 2.5 mg. of prodigiosus proteid.

After mixing, bottle in glass-stoppered bottles. Add a small piece of thymol (size of pea to 1 oz. bottle) to each bottle, and sterilize two hours at 75° C. Keep on ice.

First, a further word upon the clinical observations of accidental erysipelas in inoperable tumours. In one of my earlier papers, "The Treatment of Malignant Tumours by Repeated Inoculation of the Living Germ of Erysipelas," I collected thirty-eight cases of malignant tumours (sarcoma and carcinoma) in which an attack of erysipelas had occurred, either by accident or by inoculation. In twenty-three cases the attack was accidental, and in fifteen the result of inoculation. Seventeen were sarcoma, seventeen carcinoma, and in four cases the type of tumour was not stated. Of the seventeen cases of carcinoma, three were permanently cured. One, a probable carcinoma, was well five years after the attack of erysipelas. The remaining thirteen showed more or less temporary improvement. Of the seventeen cases of sarcoma, seven were well from one to seven years afterwards. In the remaining ten cases, nearly all showed improvement, some disappearing entirely and later recurring.

In the *American Journal of Medical Sciences*, 1906, I published six other cases of cancer—five epithelioma, and one sarcoma—in which an attack of erysipelas had intervened in the course of the disease:—

- (1) Recurrent cancer of the breast. Patient well nine years.
- (2) Sarcoma of the neck (entire disappearance). Patient well eight years.
- (3) Epithelioma of the face, eighteen years' duration (entire disappearance). Local recurrence several years later. Again disappeared under one month's treatment with the toxins. Probable recurrence six months later.
- (4) Epithelioma of the face, lip, and nose. Disappeared under very severe attack of erysipelas. Patient well two years later, when he died of another trouble.
- (5) Epithelioma of the face of two years' duration. Entire disappearance under attack of erysipelas. Patient well at last observation, six months later.
- (6) Epithelioma of nose. Eight years' duration. Entire disappearance under attack of erysipelas. Patient well several years later.

THE ACTION OF THE MIXED TOXINS UPON INOPERABLE SARCOMA.

The macroscopic as well as the microscopic changes observed by myself and others have been precisely the same as those formerly noted in cases of sarcoma treated by inoculation of the living germ. First,

the tumour becomes much paler owing to decreased vascularity; second, it becomes much more movable and less fixed to the surrounding tissues, these changes being often noted after the first two or three injections; third, it soon begins to show areas of softening, due to caseous degeneration or necrobiosis of the tumour elements; fourth, gradual disappearance, either by absorption—which is more apt to be the case in the firmer tumours (*e.g.*, spindle-cell or fibro-sarcomas)—or, in other cases (especially the round-celled and vascular varieties), by breaking down and liquefaction of the tumour-tissues. In such cases incision and drainage may be sometimes advisable, provided the tumours are in accessible regions.

These changes are precisely the same, whether the toxins have been injected directly into the tumour or whether the injections have been made in remote parts of the body, proving that the action of the toxins is systemic rather than local. In a certain number of cases—in my own experience in a little over 10 per cent.—the degenerative progress has gone on until complete absorption of the tumours has taken place and the patients have remained cured. In other cases improvement is only temporary, and after a few weeks, in spite of continued injections and increased doses, the tumour again begins to show signs of growth and continues until a fatal issue. In a very few instances, especially in cases of very large and vascular tumours, particularly the melanotic type—which of late many pathologists are inclined to class as carcinoma, instead of sarcoma—no marked beneficial effects have been noted at any time.

What is the explanation for these variable results? Why should the toxins behave so differently in these cases, causing some of the very worst and most hopeless ones to become permanently cured, and in others showing little or no effect?

The explanation is, I think, not so very difficult. My own belief, expressed sixteen years ago and held more firmly with increasing clinical experience, is that all varieties of malignant tumours are of extrinsic or microbic origin. Just what type of organism this may be—whether bacterium, protozoan, or spirochæte, or what not—is of little consequence. Assuming such origin, we have but to follow the analogy of other diseases of known germ origin. We know that in all such diseases there is a natural immunity and an acquired immunity. In the case of malignant tumours there is probably a natural immunity which is very great, but in certain cases it is absent or becomes lowered, and the germ finds a favourable site and here starts the primary malignant tumour.

The important role that trauma or injury plays in the development of

malignant tumours, now accepted by all authorities, can, to my mind, but be explained on the theory of microbic origin, some writers—*e.g.*, Tillmanns—going so far as to state that most cases of bone tuberculosis follow an antecedent injury, the bacilli, of course, being present in the circulation prior to the injury; but a naturally existing immunity or resisting power of the tissues had been sufficient, up to this time, to prevent any local infection. The injury, lowering this local resistance of the tissues, furnishes precisely the conditions favourable for the growth and development of the bacilli. Hence the origin of the tuberculous lesion.

If time permitted, I could cite many striking cases of sarcoma of the most virulent type that followed immediately upon a blow or an injury to the bone in previously perfectly healthy individuals. The aetiology of tumours is a problem far too difficult and complicated to do more than touch upon here. Assuming such extrinsic origin, the action of the toxins appears to me to produce certain changes in the blood or serum that restore the weakened or lost immunity or natural resisting power of the tissues, and the sarcoma-cell, no longer finding conditions favourable for further growth and development, undergoes a process of degeneration, with absorption in some cases and the formation of a slough in others.

The reason why a cure results in some cases is that in these the antagonistic action of the toxins is sufficient to destroy the cancer-cell and render the soil unfavourable for further growth; whereas in other cases the tumour-cells, by reason of greater vigour or better nourishment, are more resistant and, although receiving a temporary setback by reason of the changes in the blood produced by the toxins, soon accommodate themselves to the new environment and continue to grow as before.

The high temperature produced by the toxins may also be a factor in causing the improvement that follows their use. Many and repeated blood-examinations of sarcoma patients treated with the toxins show almost universally a marked leucocytosis as a result of the treatment.

That the tumour-cell is ever in a delicately balanced state is shown by the experimental investigations upon mouse-cancer during the past four years. A certain strain of carcinoma was found to grow well in Berlin mice, but would not grow at all, or in few cases only, in Danish mice of the same breed. To quote a recent and unpublished paper of Dr. Jones Ewing, "Apparent slight differences in the food supply sufficed to render the soil unfavourable to the tumour-cell. These remarkably delicate nutritional requirements of the cancer-cell suggest

that some means may be found to render the human patient's tissue unfavourable for cancer growth." And this is exactly what, in my opinion, the toxins do in sarcoma.

INDICATIONS FOR THE USE OF THE TOXINS.

While in all my earlier papers I have practically limited the use of the toxins entirely to cases of inoperable sarcoma, further experience has convinced me that they have a much wider field of usefulness. I would at present advocate their use, first, in all cases of inoperable sarcoma, except the melanotic, which are classed as of epithelial origin by many pathologists; second, in cases of sarcoma originating in the long bones in which operation means a sacrifice of the limb: if in these cases no improvement is noted at the end of two to three weeks, I would then advise excision, resection, or amputation, according to the individual case; third, in all cases of operation for primary sarcoma directly after wound healing as a prophylactic against recurrence; fourth, in addition to the foregoing, I think there is good ground for believing that the use of the mixed toxins after all operations for carcinoma would greatly lessen the number of recurrences. This opinion is based partly on the clinical observations of a considerable number of cases in which both epithelioma and carcinoma have entirely disappeared and been permanently cured from attacks of accidental erysipelas, and partly upon the marked inhibitory action of the mixed toxins upon inoperable carcinoma, as shown by actual experiments in a large number of cases.

THE USE OF THE TOXINS IN SARCOMA OF THE LONG BONES.

This deserves special mention. In a recent paper upon the "Conservative Treatment of Sarcoma of the Long Bones," read before the American Medical Association, I gave a detailed study of ninety-two cases of sarcoma of the long bones that had come under my personal observation within the last eighteen years. I stated: "The facts I have set forth are sufficient, in my judgment, to justify the giving-up of the traditional method of treating all cases of sarcoma of the long bones by immediate amputation. In most cases I believe it safe to wait for two to three weeks, the time required for a trial with the toxins, before sacrificing the leg. Sarcoma cases of extremely rapid growth will probably show little or no effect from the toxins, and one might (naturally) say valuable time had been lost by the preliminary use of the

toxins. However, it is my opinion that early operation in these cases would not have been of the slightest avail, as shown by the long, almost unbroken list of fatalities of cases treated by operation alone. On the other hand, in certain cases—probably a small number—the limb will be saved by the preliminary use of the toxins. In those in which early improvement is not marked, operation can then be performed with even greater chances of ultimate success than had the toxins not been first used.

The greatest value of the toxins in sarcoma of the long bones will, I believe, be shown to lie in a judicious combination with conservative operative treatment. By such procedure hip-joint amputation, which has been the almost uniform rule for sarcoma of the femur, will give place to an amputation below the trochanter which will leave a stump of sufficient length to permit the wearing of an artificial limb; and this is no small gain. The toxins will be administered for a considerable period of time after amputation, with the hope of destroying the cells which were left behind, and which, with operative treatment alone, cause the local and metastatic recurrences. The same rules will apply to sarcoma of the humerus.

Coming to sarcoma of the tibia, fibula, and radius and ulna, particularly of the myeloid type, in place of amputation, as formerly advised and still advocated by the great majority of surgeons, we can safely substitute either curetting or partial resection, followed by a thorough course of the mixed toxins. While good results have been obtained in a very limited number of cases in this group by operation alone, I am convinced that the number of successes will be greatly increased by combining the toxin treatment with conservative operation, as I have suggested, and my series of cases strongly supports this opinion.

As earlier diagnosis is steadily but surely coming, owing to increased knowledge of this disease, coupled with more correct interpretation of X-ray plates and the use of earlier exploratory operations, the conservative treatment along the lines I have mentioned will soon show results infinitely superior to those obtained by the radical and maiming operations thus far almost uniformly practised.

The following cases of sarcoma of the long bones are worthy of special note:—

(1) E. D., female, aged 18. Periosteal round-celled sarcoma of femur: amputation below trochanter, followed by toxin treatment for four months. Well at present, three years.

(2) C. L., female, aged 12. Periosteal round-celled sarcoma of femur: amputation below trochanter; toxins for four months after operation. Well three years and nine months.

(3) A. G., male, aged 19. Round-celled periosteal sarcoma of the femur, involving two-thirds of the shaft: hip-joint amputation advised February, 1902, but refused; developed extensive metastases in the pectoral region and a tumour the size of a child's head in the ileo-lumbar region under X-ray treatment; then the mixed toxins of erysipelas and *Bacillus prodigiosus* were given; entire disappearance. Patient well when last seen, six years later. Reported well September, 1909, seven years later.

(4) J. F., male, aged 40. Spindle-celled sarcoma of the tibia, recurrent after extirpation: preliminary use of the toxins before sacrificing the limb; entire disappearance under three months' treatment. Patient well ten years, without loss of leg.

(5) Case of Dr. A. Gerster's—Female. Round-celled myelogenous sarcoma of femur: diagnosis confirmed by Professor T. M. Prudden; too extensive for hip-joint amputation; spontaneous fracture; disappearance under four months' treatment with the toxins; bone reunited. Patient shown before the New York Surgical Society in perfect health four years later.

(6) K. K., female, aged 18. Myelogenous round- and giant-celled sarcoma of tibia: recurred very quickly after two extirpations; finally disappeared under six months' treatment with the toxins plus X-rays. Well four years.

(7) Mrs. F., aged 26. Myelogenous giant-celled sarcoma of lower end of radius: extirpation; amputation advised by Drs. Harlow and Pool: refused; toxins five weeks. Patient well at present, one year.

(8) *Sarcoma of the Femur, Periosteal; four months' duration.*—Mr. W., male, aged 40. Exploratory operation showed it to be round-celled sarcoma: amputation below trochanter, September, 1906, by Dr. Erdman; toxins for five months, under my direction, by Dr. Grausman. Patient well at present, three years later.

(9) *Sarcoma of the Femur, Periosteal; very rapid growth.*—Male, aged 10. Temperature 101° F. to 102.5° F.; exploratory operation; spindle-celled sarcoma, involving two-thirds of the shaft of the femur; toxins given in large doses for eight months; the tumour of the femur nearly disappeared. Patient died of intra-orbital or brain metastases one year later.

(10) *Periosteal Round-celled Sarcoma of the Clavicle.*—Mr. H. Very rapid growth; excision of clavicle by Dr. Maurice H. Richardson, of Boston, May, 1908. Referred to me for the toxins immediately after wound healing. Tissues about the neck suspicious of sarcomatous infiltration; immediately put upon the toxins; continued in large doses for eight months, in all eighty-seven injections; largest dose, 28 mm.; injections made into pectoral region and buttocks. Patient gained 28 lb. in weight, fully recovered his normal health, doing hard

work the last six months. Examination June, 1909, thirteen months after-ward; no trace of recurrence. (Dr. Richardson states that if the patient remains well he will attribute his recovery entirely to the toxins.)

These cases become of considerable importance when we consider that recovery from periosteal sarcoma of the femur, treated by amputation alone, is extremely rare. You are all, doubtless, familiar with Mr. Butlin's well-known work upon sarcoma of the long bones, and remember the fact that of his series of sixty-eight cases of periosteal sarcoma of the femur treated by amputation, either at the hip-joint or just below the trochanter, only one was cured.

Dr. Charles B. Nencrede, of Ann Arbor, the President of the American Surgical Association, stated recently that he had performed sixteen amputations at the hip-joint for sarcoma of the femur without a single permanent cure.

THE TOXINS AS A PROPHYLACTIC AGAINST RECURRENCE AFTER OPERATIONS FOR PRIMARY SARCOMA.

The use of the toxins as a prophylactic after operation for sarcoma I believe to offer by far the most important field of all, and one that is gradually being appreciated by the profession. At the Mount Sinai Hospital, in New York, the toxins are now used by Dr. Gerster and Dr. Lilienthal in all inoperable cases of sarcoma as well as a prophylactic after operation for primary sarcoma, and many others are beginning to use them in this way. I have already a sufficient number of cases of sarcoma in which I have used the toxins as a prophylactic measure to justify such use.

The following is a partial list of cases in which the toxins have been used after operation:—

(1) Sarcoma of finger, periosteal, round-celled (Weleh): imperfect removal; amputation considered, but a trial of toxins determined; toxins three to four months, systemic chiefly. No recurrence, nine years.

(2) Testis, round-celled (Professor Whitney): recurrent in other testis; castration; toxins after second operation for four months. Patient well at present, over four years.

(3) Testis, round-celled: very rapid growth; castration; toxins three months. Patient well over three years.

(4) Testis (round-celled sarcoma of undescended testis): removal; tumour very large; almost certain that some sarcomatous tissue was left behind; toxins as soon as wound had healed. Patient well one year later.

(5) Humerus: very large tumour, size of a man's head; amputation followed by toxins. Patient well over a year; no trace of recurrence.

(6) Clavicle: excision for rapidly growing round-celled sarcoma of clavicle (microscopical examination, Professor Whitney, Harvard): toxins begun immediately after wound was healed; suspicion of recurrence before toxins were begun; toxins given for one year. Patient in perfect health at present, one year later.

(7) Tibia, round-celled: toxins before and after amputation. No recurrence two years.

(8) Hand: four operations before toxins were given; toxins two months. Patient well six months, when lost sight of.

(9) Sarcoma of metatarsal bone, periosteal, round-celled: rapid growth; amputation; toxins four months after operation. Patient well eight years.

(10) Sarcoma of radius: amputation; toxins three months. Patient well three years.

(11) Sarcoma of femur: amputation below trochanter; toxins four months. Patient well three years.

(12) Sarcoma of femur: amputation below trochanter; toxins four months. Patient well two years and ten months.

(13) Sarcoma of femur: amputation; toxins four months. Patient well two years and nine months.

(14) Sarcoma of kidney, child aged $1\frac{1}{2}$: round-celled nephrectomy: given nearly a hopeless prognosis; toxins used almost two years. Child in perfect health at present, four years.

(15) Sarcoma of femur: amputation below trochanter; toxins six months. Patient still well at present, ten months.

(16) Sarcoma of biceps muscle, round-celled: recurred two to three weeks after excision; toxins after second operation; recurrence delayed, but not prevented; amputation shoulder-joint, January, 1908. Patient in perfect health at present, July, 1909.

(17) Sarcoma of breast: entire breast involved; rapidly growing spindle-celled sarcoma; amputation (Dr. Parker Syms); toxins two months under my direction. Recurrence about six months later; death within the year.

THE TOXINS AS A PROPHYLACTIC AGAINST RECURRENCE AFTER PRIMARY OPERATIONS FOR CARCINOMA.

While I have not sufficient data to enable me to speak emphatically in regard to carcinoma, I believe the inhibitory action of the toxins upon carcinomatous cells—sufficient in an insignificant number of cases

to cure a large inoperable tumour—to be sufficient to prevent recurrence in a considerable number of cases if used after operation. I have had one case in an inoperable epithelioma of floor of mouth and lower jaw (fig. 2). When used as a prophylactic measure in the way I have indicated, I believe the toxins to be entirely devoid of risk. No deaths have occurred in the cases in which the treatment was used as a prophylactic. In these cases the dose given is smaller, and it is not increased to the point of producing severe reactions; a moderate temperature of 99-101° F. is all that is required. This will not in any way interfere with the ordinary routine of life. The toxins can



FIG. 2.

Epithelioma of chin and lower jaw. Well seven years after treatment.

be given for long periods of time without any harmful effects, as is shown by a case of eight times recurrent sarcoma of the chest-wall in which the treatment was continued for four years, and the patient is now well, fourteen years after the beginning of the treatment, or ten years after its cessation. The treatment did not in the least interfere with his regular work. I have several other cases in which the toxins were given for two years or more without any unfavourable effects.

DURATION OF TREATMENT.

It is very hard to lay down any definite rules as to the duration of treatment that would apply to all cases. My own feeling, based upon my experience up to the present date, is that there is much more danger in stopping the toxins too soon than in giving them too long. That they can be given for very long periods without harm is shown by some of my cases, one in particular having taken the toxins with some intervals of rest for a period of nearly four years. He had had eight operations for recurrent spindle-celled sarcoma of the chest-wall. At each operation the tumour had become more malignant, and the case was considered quite hopeless from the standpoint of further operations. The treatment was given in small doses which, after the first few weeks, did not interfere with the performance of the patient's duties as a surgeon. He is to-day in perfect health, fourteen years after the beginning and ten years after the cessation of the treatment. In many of the prominently successful cases the toxins have been given for comparatively short periods—six weeks to three or four months. One, an inoperable sarcoma of the abdominal wall, had only thirty-one injections, and entirely recovered and was well when last seen, one and a half years later. Another case, an inoperable sarcoma of the abdominal wall, spindle-celled, was pronounced hopeless by Professor Maurice H. Richardson, of Harvard Medical School; diagnosis confirmed by microscopic examination by Professor Whitney; disappearance of the tumour under four months' treatment in the fall of 1894. The patient is to-day in perfect health, fifteen years later.

Another case, an inoperable sarcoma of the breast, axillary glands, and pectoral muscle; the diagnosis confirmed by Professor W. H. Welch, of Johns Hopkins; the tumour disappeared after seventy-eight injections (1895), and the patient is still in perfect health. I presented her before the Medical Society of New York in February, 1909, fourteen years later.

Another case of inoperable sarcoma of the abdominal wall and pelvis, involving the bladder, was treated with the toxins for six months (January, 1893). The tumour entirely disappeared, and I presented the patient before the New York Surgical Society in 1898 in perfect health, fifteen years later. In this case the patient had been pronounced inoperable by Dr. L. Bolton Bangs, and the diagnosis of spindle-celled sarcoma was confirmed by Dr. H. T. Brooks, Professor of Pathology at the Post-Graduate Medical School.

In a few cases there has been a recurrence of the tumour after it had once disappeared under the toxins treatment, and I feel that had the toxins been given for a longer time a cure might have resulted. My first case of sarcoma of the neck and tonsil, recurrent and inoperable, was treated with living cultures, finally resulting in a severe attack of erysipelas. The tumours nearly disappeared and the patient entirely recovered his general health, but finally died eight years later from a local recurrence.

In another case of extensive sarcoma of the back, with large metastatic tumours in the groin, the tumours entirely disappeared under living cultures of erysipelas. Recurrence quickly followed, but finally disappeared under the injections of the toxins. Patient remained well for three and a quarter years, when he died of abdominal metastases.

With a very few exceptions (four or five), all cases in which the tumours have disappeared under the toxin treatment have remained well. These few cases of recurrence furnish the most convincing evidence of the undoubted influence of the toxins upon sarcoma, inasmuch as they absolutely prove that, in these cases at least, there could have been no error of diagnosis. Can we, then, form any practical rules to guide us as to how long the toxins should be administered? I would say, give the toxins until the tumours have entirely disappeared, and then continue in smaller doses and greater intervals for three or four months longer. If no improvement is noted at the end of four or five weeks, a successful result is not likely to occur, and retardation of the growth is all that can be expected from a further use of the toxins. A concrete example may be of some help in determining the duration of treatment:—

Recurrent Inoperable Sarcoma of the Back with Metastatic Tumour of the Lower Jaw (large round-celled); entire disappearance under two months' treatment with the toxins; patient well at present, nearly two years later.—Male, aged 35; very large and rapidly-growing sarcoma of the back (lumbar region), for which he had two operations in rapid succession, in September and October, 1907. He had developed a metastatic tumour in the lower jaw, and lost 40 lb. in weight, and was sent home to die by his attending surgeon, Dr. Biddle, of the State Hospital of Pennsylvania. Microscopical examination had been made by the pathologist of the Jefferson Medical School, Philadelphia, who pronounced it a large round-celled sarcoma. The patient was brought to me in November, 1907, greatly emaciated and unable to walk without help. In view of the very rapid growth of the tumour with such early bone metastases I gave a very bad prognosis, but still thought it worth while to try the toxins. The toxins were nearly all given systemically, *i.e.*, into the buttocks; only

4 or 5 doses of the filtered toxins were injected into the tumour of the jaw. He was quite susceptible to the toxins; highest dose he ever received was 8 mm. He was not able to stand more than four or five doses a week. At the end of one month there was slight improvement; at the end of six weeks the improvement was marked; from that time on tumours melted away with great rapidity, until at the end of ten weeks there were no traces left either in the back or jaw. On February 8, 1908, three months after treatment was begun, the patient had regained his lost weight and most of his strength, and was sent home. The toxins were continued by the family physician twice a week for two months, and once a week until July, 1908, since which time he has had no further treatment. No treatment other than the toxins was given during the whole time. I presented him before the New York Orthopædic Association, February, 1908, and examined him a few weeks ago at my office. He has gained 69 lb. in weight, and there is no trace of tumour in any part of his body; he has been performing his regular duties as a manufacturer for now more than a year.

Much depends upon a judicious determination of the dosage for the given case. As a rule I like to give as much as the patient can safely stand. I always begin with one-fourth of a minim diluted with sufficient boiled water to ensure accuracy of dosage, injected either into the buttocks or pectoral region. After the individual's susceptibility has been ascertained, one can inject into the tumour itself if it is in an accessible region. The initial dose into the tumour should always be less, not more, than one-fourth of that used elsewhere. I believe it a good plan to give the injections alternately into the tumour and into the buttocks. Daily injections should be given, increasing by one-fourth of a minim until the desired reaction—namely, a temperature of 102-104° F.—has been obtained. This should be modified to suit patients in a weakened condition. Having secured the desired reaction, the dose should no longer be increased until it fails to give a reaction, when it can again be increased by one-fourth to half a minim. The dose varies greatly with different individuals; the highest dose ever given in many of the cured cases has been 7 mm. to 8 mm. On the other hand, the case of spindle-celled sarcoma of the sternum and cervical glands (microscopic examination by Dr. James Ewing, Professor of Pathology, Cornell University Medical School) showed little improvement until large doses, as high as 30m, had been given directly into the tumour. The treatment was carried out by another physician, Dr. David John, of Yonkers, New York, under my direction. The tumours entirely disappeared, and the patient is still well, nearly three years later. I presented her before the New York Surgical Society a year ago. I have at present under

treatment at the Hospital for Ruptured and Crippled at New York a little girl, aged 6, with a three-times recurrent sarcoma of the face, in which the dose was carried up to 20 m, injected directly into the tumour, before a marked reaction was obtained. She has now taken the toxins for nearly four months, and under these large doses has shown very marked improvement. When I left the tumour had nearly disappeared, and I am hopeful of a cure. On several occasions the toxins were stopped for a few days, and each time there was a rapid increase in growth. I have never seen a case in which the inhibitory action of the toxins upon sarcoma has been more beautifully demonstrated, whether a cure result or not.

The greatest obstacles to a fair trial of the toxins up to the present time have been: (1) The difficulty of obtaining a preparation of the toxins of uniform standard of strength and efficiency. I will here state that all of my results have been obtained from toxins prepared by Dr. B. H. Buxton, Professor of Experimental Pathology, Cornell Medical School, from 1893 to 1906; from 1906 to the present time by Dr. Martha Tracy, Assistant Pathologist to the Huntington Cancer Research Fund, who received personal instructions from Dr. Buxton in the method of preparing the toxins. Dr. Tracy's own modification of the method of preparation, by means of which it has been possible to standardize a dose, is a marked step in advance. The preparations before this time were much more variable in strength and efficiency. (2) The published failures of a few men who have given the method a limited and most imperfect trial, usually with a preparation of the toxins entirely different from that used by myself. (3) The number of cases of sarcoma is so limited that the ordinary physician or surgeon sees not more than one or two cases a year; even in large hospitals there may not be seen more than five or six cases a year, and these are chiefly operable cases. In these the tumour is removed, patient is sent home until later, when a recurrence has taken place, he is again sent to the hospital. If operable, the tumour is again removed; if not, he is sent home and his physician advised to make his end as comfortable as possible.

Occasionally the physician has heard of the use of the mixed toxins in such cases; if he has read of the method, he has probably forgotten the details and has little faith in its value. Now and then a surgeon is found who takes the time to give the matter special thought, and who is willing to give the patient the benefit of a trial with the toxins. He begins with some fears and more doubts, and when he sees the patient in one of the severe chills, sometimes with marked cyanosis, very rapid

and feeble heart action, with a temperature of 103-5° F., he hesitates to go on, or is unable to instil into the patient the courage and confidence so necessary to enable him to continue the treatment to a successful termination. For these or other reasons injections are stopped and the treatment pronounced a failure.

DANGERS OF THE TOXINS.

The risks of the treatment have been unduly emphasized. While I have personally found the administration of the mixed toxins practically free from danger, there have been several fatal cases in the hands of other physicians brought to my notice which, taken together, show that there are certain risks connected with the treatment. If, however, the precautions which I have always carefully emphasized in former papers be observed, these risks, I believe, will be reduced to a minimum. Most of the fatal cases that have occurred have been due to a neglect of these precautions. In my own experience in nearly 500 cases there have been only three deaths.

In the majority of the fatal cases thus far observed death was apparently due to an embolism. This was the cause in two of my own cases, and in both instances the general condition of the patients was extremely bad; there was generalization of the disease and very marked involvement of the mediastinal glands. In both cases the patients had only very small doses of the toxins, which were not pushed to the point of giving any marked reactions.

Most of the deaths in the hands of other men have been due, I believe, to too large an initial dose given directly into a vascular primary growth. In recent years I no longer inject the initial dose directly into a tumour, but first test the individual susceptibility of the patient by systemic injections in the buttocks or pectoral region; after a few such injections local treatment may be begun, always starting with a minimum dose. I rarely give more than $\frac{1}{8}$ mm. into the tumour in children, especially if situated in the neck or mediastinum, and never more than $\frac{1}{4}$ mm. in adults. I have seen a temperature of 105° F. result from $\frac{1}{8}$ mm. injected into a large cervical tumour in a child. In one of the fatal cases in the hands of other men death resulted from an injection of $\frac{1}{4}$ mm. of Dr. Tracy's early preparation (twice as strong as at present) directly into a mediastinal tumour. This was an elderly woman of very feeble vitality.

Another case, recently brought to my notice by the physician in charge, contains an important lesson—*i.e.*, that the toxin should never be administered by any physician who is not willing to give sufficient time, I will not say to thoroughly read all of the literature connected with the treatment, but at least the directions upon the bottle. In this case the initial dose was 5 mm. injected into a vascular tumour. The physician stated that he had turned the case over to his assistant, who gave him 5 mm., and the patient died in fifteen minutes. Death might also have resulted had he given him 20 gr. of morphine, though perhaps not so quickly. The toxins are, indeed, like strychnine and morphine, safe if judiciously and intelligently administered, but exceedingly dangerous if given in the way described.

I have known of another instance, a case of sarcoma of the lower jaw, in which—after two operations had been performed and it was not thought that the disease had been entirely removed—the toxins were advised immediately after operation. In this case, also, the treatment was turned over to an assistant. Although never within the last fifteen years have the directions sent out with the toxins called for larger initial doses than $\frac{1}{4}$ mm. or $\frac{1}{2}$ mm., the assistant in this instance proceeded to give 20 mm. as a first dose. This was some years ago, when the preparation was considerably weaker than at present; at least its strength was more variable. Although the patient went into coma and remained comatose and delirious for some time, he finally recovered and is well now, two years afterwards, the tumour of the jaw having been cured.

In a few other cases, however, in which death resulted, the ordinary precautions had apparently been carried out. One such case was seen by myself in consultation, and the treatment advised and a general outline of the dosage given. This was a woman, aged 55, with a very large, vascular sarcoma of the ilium. Her general vitality was much impaired and circulation not good. The toxins were begun in minute doses, $\frac{1}{4}$ mm., and gradually increased up to the point of reaching a temperature of 103-4° F. After about two weeks' treatment, an injection of 12 mm. caused a severe chill; the patient became very weak and exhausted, and did not recover. In this case, had I had personal charge of it, I have no doubt that, seeing that she did not bear the toxins well and that the temperature remained high, I should not have increased the dose as rapidly as was done.

Each case must be treated on its individual merits. In one case death resulted from increasing materially a dose which, the day before,

had already produced a severe reaction, the patient still having a temperature of 102° F. at the time the injection was given. It is a very good rule not to repeat injections as long as the patient has any material rise of temperature, but to wait until the latter has fallen to normal or nearly normal. After the desired reaction is obtained, causing a temperature of 102-3-4° F., according to the vitality of the patient, one should not increase the dosage until it has failed to produce such reaction. I have never known of a fatality from the use of the toxins as a prophylactic after operation, nor of any dangerous symptoms.

FINAL RESULTS.

Up to the present time I have had fifty-two cases of inoperable sarcoma successfully treated with the mixed toxins of erysipelas and *Bacillus prodigiosus*. Of these, thirty-five have remained well from three and a quarter to sixteen years; twenty-eight from five to sixteen years, and fourteen from ten to sixteen years. To the thirty-six successful cases published in the *American Journal of the Medical Sciences*, in March, 1906, I have been able to add sixteen others.

In the first thirty-six cases reported in 1906 there was, in addition to the tumours being adjudged inoperable by leading surgeons, a careful microscopical diagnosis made in all but two instances. One of these cases was a large tumour of the sacrum, pronounced sarcoma and inoperable by the attending physicians and surgeons of St. Luke's Hospital of New York. The patient had lost 40 lb. in weight and could not walk without assistance. The tumour entirely disappeared under two months' treatment with the mixed toxins; in three months he had regained his normal weight and resumed his work. He is well at the present time, fourteen years later. The other case was a large inoperable tumour of the right iliac fossa, highly vascular, pronounced sarcoma by Dr. G. R. Fowler, of Brooklyn, after an exploratory laparotomy. The tumour disappeared under the toxin treatment, and the patient remained well for eight years, when he died of another disease.

The majority of these cases have been shown from time to time before the New York Surgical Society, one of them sixteen years after treatment. At the Hartford Medical Society last year I showed three cases, all residents of Hartford. One, an enormous sarcoma of the gluteal region, well for fourteen years, was recurrent and had been

pronounced inoperable by Dr. Charles McBurney when I began the treatment. The second case was a spindle-celled sarcoma of the breast and pectoral region; diagnosis confirmed by Dr. W. H. Welch, of Johns Hopkins Medical School; treated under my direction at Hartford; well fourteen years. The third case was a round-celled sarcoma of the tonsil and larynx, with metastases in both cervical regions. Entire disappearance under six months' treatment; well at present, nearly three years afterwards; diagnosis confirmed by microscopical examination, Cornell Medical Schools Laboratory.

In my paper of April, 1906, *American Journal of the Medical Sciences*, I tabulated the successful cases of other men, sixty in number. Since that time, from personal communications and published reports, this number has greatly increased, until now I believe that considerably more than 100 cases have been successfully treated by other surgeons. I am much gratified to find two successful cases, recently reported in the *Lancet*, March 20 and May 22, 1909.

I cannot give in detail all of my successful cases. This would require too much space, and the majority have already been reported in my paper published in the *American Journal of the Medical Sciences*, March, 1906, and in the *Annals of Surgery*—Transactions of the New York Surgical Society—since that date. I believe, however, that it may be of sufficient interest to give a brief history of a few of these cases, together with a brief abstract of the entire number:—

(1) *Inoperable Spindle-celled Sarcoma of the Abdominal Wall; entire disappearance under two and a half months' treatment with the filtered and unfiltered toxins; patient perfectly well at present, fourteen years afterward.*—Upon this patient exploratory laparotomy was performed in August, 1893, at the Massachusetts General Hospital, by Dr. Maurice H. Richardson, now Professor of Surgery at the Harvard Medical School. The tumour was found to involve such a large portion of the abdominal wall that removal was quite impossible. A portion was excised and examined by Dr. W. F. Whitney, pathologist of the Massachusetts General Hospital, who pronounced it spindle-celled sarcoma. The patient was referred to me by Dr. Richardson in October, 1893, and immediately put upon the injections of the mixed toxins. After her return home, two and a half months later, she had no further treatment, and she has remained perfectly well up to the present time.

(2) *Spindle-celled Sarcoma of the Scapular Region, involving a large part of the left half of the Thoracic Wall.* (Diagnosis confirmed by microscopic examination made by Dr. H. T. Brooks, Professor of Pathology at the Post-graduate Hospital.)—The patient was admitted to the New York Cancer Hospital on January 20, 1894. The tumour entirely disappeared under three

months' injections with the mixed unfiltered toxins, by absorption, without any breaking down. The patient was in good health when last heard from, in 1904, ten years later. (Figs. 3, 4, 5.)

(3) *Inoperable Recurrent Angiosarcoma of the Breast, treated with the Mixed Toxins of Erysipelas and Bacillus prodigiosus.*—The patient, aged 59, had a three-times recurrent sarcoma of the left breast of such size that operation was entirely out of question (fig. 6). The patient was referred to me by Dr. G. F. Shrady, of New York, as an inoperable case. She was admitted to the



FIG. 3.

Spindle-celled sarcoma of scapula and chest-wall. Tumour 13 in. vertically behind, 7 in. vertically in front, adherent to chest wall. Arm could only be raised to right angle. Photograph taken before treatment.

New York Cancer Hospital, January 20, 1895, and placed in the incurable ward. The diagnosis was confirmed by microscopic examination made by Professor T. M. Prudden, of the Medical Department of Columbia University, who pronounced it a round-celled angiosarcoma. Under the toxin treatment the patient recovered and remained well until 1903, eight years afterwards, when she

fell downstairs and received an injury which proved fatal. She was examined at this time by my associate, Dr. W. A. Downes, who found no trace of a recurrence. (Fig. 7.)

(4) *Mixed-celled Sarcoma of the Parotid, three times recurrent, treated with the Mixed Toxins for six months: patient perfectly well, eleven years later.*—The patient had been previously operated upon by Dr. William T. Bull, who



FIG. 4.

Showing entire recovery. Patient well twelve years after treatment.

referred her to me for the toxin treatment in 1895, as there was no hope of benefit from further operation. She was treated for four months. The tumour slowly decreased in size until there was only a very small freely movable nodule left, about the size of a hazel-nut. This was excised, and careful microscopic examination showed no trace whatever remaining of sarcoma. The patient remains in perfect health at the present time, eleven years later.

(5) *Spindle-celled Sarcoma of the Iliac Fossa, probably involving the Ilium.*—Mrs. D., aged 40, noticed pain in the right iliac fossa in the early part of 1895, and in May discovered a tumour in this region. This steadily increased in size, and on October 19, 1895, Dr. Johnston, of Boston, performed an exploratory laparotomy. His description of the condition found is as follows: "A tumour was found in the right iliac fossa about the size of a cocoanut, attached to the ilium as well as to the abdominal wall, and totally inoperable.



FIG. 5.

Back view of patient shown in fig. 4.

Its exact point of origin could not be made out, but from the exploration and subsequent examination I am convinced that it started from the inner portion of the crest of the ilium." A part of the tumour was excised and examined by Dr. William F. Whitney, pathologist to the Massachusetts General Hospital and Instructor in Pathology in the Harvard Medical School, who pronounced it spindle-celled sarcoma. In November, 1895, Dr. F. Cobb, of Boston, started the injections with the mixed unfiltered toxins, and at the end of six weeks, as

Dr. Cobb states, the growth had entirely disappeared. In the spring of 1896, however, the patient developed a local recurrence, and on May 17, 1896, she was sent to me for treatment, and was admitted to the New York Cancer Hospital. At this time examination showed a hard mass on the right side extending from the crest of the ilium nearly to the umbilicus and as far to the left as the median line. The tumour was apparently located in the ilium and abdominal wall. The toxins were given, with occasional intervals of rest, for three months, during which time the tumour had decreased to one-fifth of its original size. I then gave her a rest of two months, when she was readmitted to the hospital. During this interval the tumour had increased considerably



FIG. 6.

Recurrent angio-sarcoma (round-celled).

in size, but again began to yield immediately after renewed treatment. She remained under treatment until June, 1897, at which time the tumour had almost entirely disappeared, and examination a few months later showed no trace of it left. I made a personal examination of the patient in the latter part of 1907, at which time she was perfectly well, more than ten years after the cessation of the treatment. During this entire time she led a very active life, supporting a large family of children. Careful palpation failed to show any evidence whatever of the tumour.

(6) *Round-celled Sub-periosteal Sarcoma of the Femur, involving lower two-thirds of the Shaft, with extensive Metastases.*—A. G., aged 19. A tumour in the lower portion of the femur was first noticed in November, 1901. There was no history of trauma. This tumour gradually increased in size, and was accompanied by loss of weight and deterioration of general health. The patient was referred to me on February 5, 1902, by Dr. W. R. Townsend, of the Hospital for Ruptured and Crippled. Physical examination at that time showed a large tumour occupying the entire lower two-thirds of the left femur, fusiform in shape, and most prominent in the region of the condyles. On the



FIG. 7.

Disappearance of tumour under toxins. Patient well eight years later.

outer aspect of the thigh, about $1\frac{1}{2}$ in. above the joint, there was a soft fluctuating area. There was slight impairment of the functions of the joint, but the joint itself was not involved. An incision was made under ether anæsthesia over the fluctuating area, and 3 oz. of clear serum, similar to that found in sarcoma of the bone which has undergone cystic degeneration, was evacuated. By means of a curette a considerable portion of typically sarcomatous tissue was removed. This was examined microscopically by Drs. E. K.

Dunham, of Bellevue Hospital, and B. H. Burton, of Cornell University, and pronounced small, round-celled sarcoma. The patient absolutely refused amputation at the hip-joint, which I strongly urged. I was at this time just beginning to try the X-ray treatment of inoperable malignant tumours, and gave the patient four exposures a week. At the end of one month the tumour had decreased in size 1 in. The treatment was continued during the entire summer and fall of 1902. The patient gained considerably in weight, but in December, 1902, developed a metastatic tumour in the left pectoral region. This grew very rapidly, and when it had reached the size and thickness of the hand, I removed it with scissors and curette, under ether anaesthesia. Shortly after this, a large tumour, about the size of a child's head, developed in the ilio-lumbar region on the right side; it filled up the whole iliac fossa and extended up to the ribs. I then put the patient upon large doses of the mixed toxins of erysipelas and *Bacillus prodigiosus*. After about four weeks the tumour in the ilio-lumbar region began to soften and break down. As soon as fluctuation became distinct, I made a posterior opening and evacuated a large amount of necrotic tumour-tissue. A tube was kept in place and the sinus drained for about a year. No X-ray treatment was applied to the ilio-lumbar tumour. The sinus in the leg has persisted up to the present time; examinations of several curettings have failed to show any evidence of sarcoma. At the present time, over seven years from the beginning of the treatment, the patient is apparently in perfect health, and there is no longer any evidence of sarcoma to be found.

(7) *Myelo-Sarcoma of the Lower End of the Tibia, twice recurrent; disappearance under eight months' treatment with the mixed toxins.*—K. K., female, aged 21. Admitted to the Hospital for Ruptured and Crippled, September, 1904; operated upon October 11, by Dr. V. P. Gibney. The entire lower third of the tibia was involved, only a thin outer shell being left, as shown by X-ray photographs. A second operation was done January 5, 1905, consisting in the removal of a large mass of sarcomatous material with chisel and curette. At neither operation was any attempt made to remove the entire tumour. After the second operation the patient was put upon the mixed toxins and, a few weeks later, the X-rays were given in addition to the toxins. At first there was some increase in size, but later, under large injections, followed by more severe reactions, the tumour was held in check and, finally, slowly receded. The treatment was continued until July, 1905, by which time the tumour seemed to have disappeared entirely. The patient has remained in perfect health, without any sign of recurrence, up to the present time, four and a half years later. The microscopical examination in this case was made by Dr. F. M. Jeffries, Professor of Pathology to the New York Polyclinic. The patient was shown in perfect health before the Medical Association of Greater New York, February 15, 1909, and is well at present, September 1, 1909.

(8) *Spindle-celled Sarcoma of the Sternum.*—Mrs. G., aged 38. Mother died of tumour of brain twelve years ago. Past history: In June, 1906, first noticed

enlargement of upper portion of sternum, especially marked over sterno-clavicular joint on the right side. This slowly increased in size, and in December, 1906, I was called to see the patient in consultation with Dr. John, of Yonkers. I advised an exploratory operation to confirm the clinical diagnosis of sarcoma. This was performed on December 29, 1906, and the specimen removed was examined by Dr. James Ewing, Professor of Pathology of Cornell University Medical School, and Drs. B. H. Buxton and Martha Tracy, of the Loomis Laboratory, who pronounced it spindle-celled sarcoma. On January 6, 1907, the mixed toxins were begun by Dr. John under my direction. The treatment was given every other day in gradually-increasing doses; by the end of January the dose had reached $2\frac{1}{2}$ mm., which was followed by a temperature of 103.4° F. After twenty injections had been given the tumour had diminished considerably in size, and the treatment was suspended for two weeks. At the end of this time examination showed the tumour to have increased again, and a small lump was observed beneath the sterno-mastoid muscle. The injections were continued at varying intervals for six months until June, 1907, the highest dose given being 30 mm. injected directly into the tumour. The tumours slowly decreased in size, even after the cessation of the treatment, and finally disappeared, and the patient at present—August 10, 1909—has had no recurrence. Her physician, Dr. John, writes me, August 10, 1909, that she suffers from periodic headaches, and has fallen on one or two occasions. This may mean brain metastasis, or it may be an independent condition.

(9) *Sarcoma of the Ilium*.—N. G., male, aged 13. Family history, good. Past history: Fell downstairs two years ago, striking left pelvic region; in bed about two weeks; fell again a year later, striking same side; laid up in bed for three weeks. Soon noticed some swelling in region of left ilium, which gradually increased in size; during last six months there has been some slight loss of flesh and decline in general health. Patient referred to me by Dr. La Ferté, of Detroit, in August, 1906. Physical examination at this time showed a tumour involving almost the entire left ilium, being hard in some and soft in other places; considerable limitation of motion in the left leg, marked loss of flexion of left thigh; has had some pain during last year, which at times was paroxysmal. The patient was admitted to the General Memorial Hospital in August, 1906, and put upon the mixed toxins; the treatment was continued for a little over a year, with two intervals of a few weeks' rest; highest dose, 8 mm. The injections were made into the buttocks rather than into the tumour. The swelling gradually decreased and limitation of motion nearly disappeared. At the end of a year the tumour had practically disappeared, although there remained some enlargement, apparently due to the new-bone formation, as often seen in osteo-sarcoma. I examined the patient in June, 1909, and he remains at present in perfect health, three years later.

(10) One of the most remarkable results yet obtained was a case of inoperable round-celled sarcoma of the upper jaw with metastases, successfully treated by Dr. O. K. Wineberg, of Lake Park, Minn., published in the *Medical Record* of May 3, 1902. The patient was so desperately ill at the beginning

of the treatment that no photograph could be taken until he had had sixty-three injections and the tumour had greatly improved, having diminished to one-half its original size (figs. 8, 9). At the time the treatment was begun the man weighed 113 lb.; he was jaundiced, cachectic, and the abdomen markedly swollen; pulse 165, weak and irregular; he was not expected to live but a few days. The important feature of this case is that all the injections were given systemically, either into the abdominal wall or into the arm. At the end of three weeks the jaundice disappeared, as also the metastases in the axillary region; there was marked decrease in the size of the tumour of the jaw; the



FIG. 8.

Round-celled sarcoma of upper jaw, with abdominal metastases. Photograph after sixty-three injections; tumour had diminished to about half original size.

toxins were given in as large doses as the patient could bear, from August, 1901, to January, 1902—103 injections in all. In three weeks he had gained 11 lb.; in four weeks he resumed his work as veterinary surgeon. Four months after treatment he made a trip to New York to show me the result, and I presented him before the Surgical Section of the New York Academy of Medicine. The diagnosis in this case was further confirmed by microscopical examination made by Dr. William H. Welch, of Johns Hopkins University, and Dr.

James Ewing, Professor of Pathology at Cornell University. The patient had remained in perfect health six years later.

(11) *Inoperable Round-celled Sarcoma of the Ovary; Inoperable Tumour made Operable by Toxin Treatment.*—Mrs. E., aged 32. Exploratory laparotomy November, 1904, revealed a large immovable tumour filling up the whole pelvis and extending above the umbilicus. Tumour was exceedingly vascular. A portion of the specimen was removed for the pathological report of the Government Laboratory at Manila. This report read as follows: "An apparently rapidly growing and infiltrating, very cellular growth of probably perithelial origin; perithelial hemangiosarcoma." The patient was admitted to

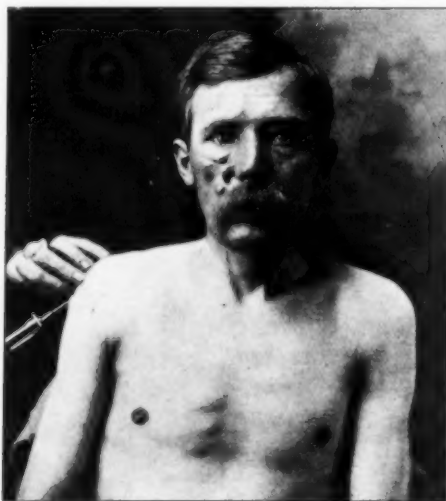


FIG. 9.

Photograph taken after three months' treatment; tumour had diminished to one-third original size. Patient well nine years later.

the General Memorial Hospital in the service of Dr. Henry C. Coe, attending gynecologist to the General Memorial Hospital, February 20, 1905. The opinion of Dr. Coe and the others who saw her was that the tumour was clearly inoperable. The tumour, which filled up the entire lower abdomen, extending above the umbilicus, was firmly fixed. The case was turned over to me by Dr. Coe for treatment with the toxins, which were given from February 24 to May 31, forty-seven injections in all. In addition to the toxins, the patient had twenty-two X-ray exposures. There was a very slow diminution in the size of the tumour, with marked increase in mobility, so much

so that it was thought possible that the tumour could be removed by another operation. Operation performed June 12, 1905, by Dr. Coe. A tumour the size of a child's head was found, originating in the right ovary. It was entirely free from adhesions. No metastases were found. The tumour was very easily removed, the entire operation not taking longer than fifteen minutes. The effect of the treatment was very good, as shown by the pathological report by Dr. Tracy: "The tumour within was very much degenerated, the contents being almost of a puriform consistence. Therefore it was difficult to fix and stain, and the diagnosis is not clear. Dr. Ewing is unwilling to say whether it is sarcoma, endothelioma, or carcinoma, though of the malignancy there is no doubt."

The report of a less degenerated portion of the tumour by Dr. Clark, the pathologist to the General Memorial Hospital, was round-celled sarcoma, which corresponded with reports of specimens removed before treatment. The patient made an uninterrupted recovery, gaining 28lb. in weight within six months, and



FIG. 10.

Inoperable sarcoma of sacrum. Treatment for six months. Patient well fourteen years later. (See Case 10, p. 37).

returned home in perfect health. Within six months she conceived, and gave birth to a healthy child in summer, 1906. She remained well up to the latter part of January, 1907, when she contracted pneumonia, and died within a few days. While the patient had not remained sufficiently well to be classed as a cure, complete restoration to health and prolongation of life for nearly two years make it worthy of note. It is possible that the supposed attack of pneumonia may have been due to metastases in the lungs. The chief interest in the case lies in the fact that an inoperable and very vascular sarcoma of the ovary, under a three months' treatment with the toxins, was transformed into a degenerated mass of almost puriform consistence, easily removable by operation.

Table of Personal Cases of Complete or Partial Success, with Late Results.¹

No.	Age	Sex	Date	Locality	Type of tumour	Treatment, duration	Result (immediate)	Result (final)
1	35	M.	May, 1891	Neck and tonsil, recurrent, inoperable	Spindle-celled	Repeated injections of living cultures, four months; one attack of erysipelas	Tumour nearly disappeared, general health restored	Patient lived eight years, and then died of recurrence
2	40	M.	April, 1892	Back and groin, recurrence, inoperable	Mixed-celled (round and oval)	Mixed cultures of erysipelas; four attacks of erysipelas; finally toxins	Entire disappearance (vide text)	Patient remained well three and a half years, then died of abdominal metastasis
3	17	M.	January, 1893	Abdominal wall and pelvis, inoperable	Spindle-celled	Mixed toxins (filtered); erysipelas and <i>Bacillus prodigiosus</i>	Entire disappearance in four months treatment	Patient in perfect health sixteen years later
4	29	F.	October, 1893	Abdominal wall, inoperable	Spindle-celled	Mixed toxins three months (Buxton)	Entire disappearance	Patient in perfect health at present, sixteen years later
5	17	F.	January, 1894	Leg, popliteal and space, recurrent; osteo-sarcoma of foot	Spindle-celled	Mixed toxins (Buxton)	Entire disappearance, recurred one and a half years; amputation below trochanter, recurred in gluteal region; finally disappeared under continued toxin treatment	Patient well in December, 1905, twelve years from beginning of treatment, ten years since end
6	34	F.	June, 1894	Chin, lower jaw, floor of mouth, inoperable	Epithelioma (microscopic examination)	Mixed toxins three months (Buxton)	Entire disappearance	Patient well when last heard from, six years later
7	16	F.	June, 1894	Chest-wall, very extensive, inoperable	Spindle-celled	Mixed toxins four months (Buxton)	Entire disappearance	Patient well, 1907, thirteen years later
8	24	M.	March, 1904	Chondrosarcoma of ilium, very large	Chondrosarcoma	Mixed toxins (Buxton)	Entire disappearance	Recurred seven months later and finally proved fatal
9	24	F.	October, 1894	Sarcoma of omentum, mesentery, and gall-bladder	Round-celled	Mixed toxins five months (Buxton)	Entire disappearance	Patient well, January, 1908, fourteen years

10	38	M.	March, 1895	Sacrum, inoperable	No microscopic examination, but inoperable tumour of sacrum examined by several surgeons	Mixed toxins six months (Buxton)	Entire disappearance in three months; patient gained 40 lb. in weight	Patient well, January, 1906, twelve years later
11	30	F.	1893	Gluteal region, inoperable, recurrent	Spindle-celled	Mixed toxins three months (Buxton)	Entire disappearance	Patient in good health, no recurrence, December, 1905, twelve years later
12	40	F.	Nov., 1895	Intra-abdominal, inoperable, size of child's head	Spindle-celled	Mixed toxins, three periods of about two months each	Tumour disappeared, and again disappeared under further treatment	Patient in perfect health, January, 1909, fourteen years later
13	16	M.	October, 1893	Sarcoma of pharynx, inoperable	Spindle-celled	Mixed toxins (unfiltered) eight months (Buxton)	Entire disappearance	Patient well when last heard from, six years later
14	20	F.	Feb. 18, 1896	Hand	Spindle-celled	Mixed toxins eleven months (Buxton)	Entire disappearance in two months	Recurred two years later, yielded for a time to further treatment, finally grew rapidly and proved fatal in another year
15	55	M.	1893	Iliac fossa	Round-celled	Mixed toxins two months (Buxton)	Entire disappearance	Patient well one and a half years later, when she returned to Germany
16	18	F.	1897	Abdominal wall	Spindle-celled	Mixed toxins, thirty-one injections (Buxton)	Entire disappearance	Patient well over eight years, no recurrence
17	59	F.	1895	Breast, recurrent, inoperable	Round-celled, angio-sarcoma	Mixed toxins six months (Buxton)	Entire disappearance with exception of small nodule, which was removed for pathological examination	Patient in perfect health, September, 1909, fifteen years later
18	37	M.	1894	Chest-wall, eight times recurrent	Spindle-celled, very vascular	Mixed toxins (filtered and unfiltered), (Buxton)	Disappearance, two or three small nodules removed in first three years	Patient well twelve years later
19	5	F.	1897	Sarcoma of lower lip, three times recurrent	Small round-celled	Mixed toxins six weeks (Buxton)	Entire disappearance	

¹ From *Amer. Journ. Med. Sci., Philad.*, 1906, N.S., cxxxi, pp. 375-430. Corrected to date, September, 1909.

No.	Age	Sex	Date	Locality	Type of tumour	Treatment, duration	Result (immediate)	Result (final)
20	32	F.	1897	Sarcoma of parotid, three times recurrent	Mixed-celled	Mixed toxins six months (Buxton)	Disappearance except small nodule excised, no longer traces of sarcoma	Patient in perfect health, with no recurrence, Jan. 1908, eleven years
21	41	M.	1897	Sarcoma of parotid, recurrence, inoperable	Spindle-celled	Mixed toxins four months (Buxton)	Entire disappearance	Patient free from recurrence six years afterward, when he suddenly died of gastric hemorrhages
22	35	M.	Feb., 1898	Tibia, recurrent, inoperable except by amputation of thigh	Spindle-celled	Mixed toxins three months (Buxton)	Entire disappearance	Patient well at present, July, 1909, eleven years later
23	16	M.	1897	* Very large and vascular	No microscopic examination, exploratory laparotomy (Dr. G. R. Fowler, New York)	Mixed toxins several months	Entire disappearance	Patient well eight years later, when he died of other trouble
24	44	F.	1894	Sarcoma of spine, inoperable, recurrent	Spindle-celled	Mixed toxins two months	Entire disappearance	Patient reported well eight years later
25	52	M.	October, 1900	Parotid, recurrent	Round-celled, microscopic examination by Prof. W. H. Welch of Johns Hopkins	Mixed toxins nine months	Entire disappearance	Patient in perfect health six years, and then died of other trouble
26	35	M.	1902	Pectoral region and axilla, inoperable, size of two fists	Round-celled	Mixed toxins three months, later toxins and X-ray	Nearly disappeared before X-rays began, later all disappeared	Recurred one year later, again disappeared; recurred and finally proved fatal, September, 1905
27	8	M.	1902	Back, recurred three times	Small round-celled	X-ray at first failed, mixed toxins later	Entire disappearance	Patient well, no recurrence September 1, 1909, seven years
28	38	F.	May, 1901	Abdominal wall, involving bladder wall	Fibro-sarcoma	Mixed toxins four months	Entire disappearance	Patient perfectly well January, 1908, seven years

29	16	M.	Feb., 1902	Sarcoma of femur, lower two-thirds, with extensive multiple metastasis	Small round-celled	X-rays eight months for primary tumour, metastasis developed in pectoral region and iliac fossa, mixed toxins nearly a year	Entire disappearance	Patient apparently free from sarcoma in any region, September 1, 1909, seven years
30	20	M.	Feb., 1902	Spine, dorsal, very large	Round-celled, paralysis of bladder and rectum and lower extremities, loss of 60 lb. weight	Mixed toxins (Buxton) four months	Entire disappearance	Patient able to walk without crutches in eight months, and without cane in one year; perfect health at present, seven years later
31	56	M.	Feb., 1904	Chest, three times recurrent	Spindle-celled	Mixed toxins (Buxton and Parke, Davis)	Partially removed by operation, disappeared entirely	Patient well without recurrence nearly two years
32	16	M.	May, 1904	Chest-wall, ribs, and pleura	Round-celled, very vascular	Mixed toxins and X-ray (Buxton and Parke, Davis)	Entire disappearance in six months	Recurred in seven months and increased in spite of renewed treatment; death October, 1905
33	28	F.	October, 1903	Intra-abdominal involving mesentery, size of child's head	Small round-celled, examined by Prof. W. F. Whitney, Harvard Medical School	Mixed toxins (Buxton and Parke, Davis) with X-ray	Entire disappearance, sudden high temperature with signs of peritonitis, development of faecal fistula, recovery	Small mass felt other side of abdomen one year later (died within six months)
34	34	M.	October, 1905	Tonsil and neck, recurrent, size of half orange	Small round-celled	Mixed toxins (Buxton), toxins eight months old, infection local and in pectoral region	Entire disappearance in six weeks, X-ray and radium previously tried and failed	Patient well at present, September, 1909, four years
35 ¹	42	F.	1895	Breast and pectoral region, inoperable	Spindle-celled, microscopic examination confirmed by Prof. W. H. Welch, of Johns Hopkins	Mixed toxins (Buxton), seventy-eight injections (local)	Entire disappearance	Patient well August, 1909, fourteen years later
36	18	F.	1905	Tibia, recurrent twice	Round-celled	Mixed toxins five months, with X-rays part of time	Tumour disappeared	A small ulcer remained at last observation, July, 1909, four years

¹ In this case the treatment was carried out under my frequent direction by Drs. Storrs and Griswold, of Hartford, Conn.

Brief Report of Successful Cases not included in the Tabulated Report of the 1906 Paper¹:—

(1) *Three-times Recurrent Spindle-celled Sarcoma of Cheek.*—Q., male, aged 50. Mixed toxins; entire disappearance under two months' treatment. Perfect health August, 1909, five years later.

(2) *Large, Recurrent, Inoperable Tumour of Pelvis* (pronounced sarcoma by Dr. Stannard).—M. L., female, aged 50. Operation at the Post-Graduate Hospital in 1894; impossible to remove tumour. Portion received for microscopical examination. Marked decrease in size under four months' treatment with the mixed toxins; then a second futile attempt to remove the growth was made by Dr. Coe and myself. The tumour slowly disappeared without further treatment. Patient shown before the Medical Society of Greater New York in February, 1909, by myself. Perfectly well fifteen years later.

(3) *Recurrent, Inoperable Round-celled Sarcoma of the Neck* (diagnosis confirmed by microscopical examination made by Dr. W. R. Steiner, pathologist to the Hartford Hospital).—A. P., female, aged 2 years 10 months. Tumour recurred two weeks after first operation; again removed; microscopical examination again showed typical round-celled sarcoma; entire neck involved, from mastoid to clavicle. In March, 1902, the toxin treatment was begun and carried out by Dr. M. McKnight, of Hartford, Connecticut, under my direction for six weeks. The tumour entirely disappeared, and the patient is perfectly well at present, seven years later.

(4) *Very extensive Round-celled Sarcoma of the Back, Lumbar Region, twice Recurrent within a few weeks.*—C. E. C., male, aged 30. Metastases in the lower jaw; marked emaciation; extreme weakness; entire disappearance under two and a half months' treatment with the toxins. Patient well at present, nearly two years; gained 69 lb. in weight.

(5) *Primary Sarcoma of the Tonsil involving Pharynx, nearly blocking up Fauces; Metastases in the Neck on both sides; pronounced Inoperable by half a dozen physicians.*—A. L., female, aged 11. Treatment with the mixed toxins begun November, 1906, continued with intervals of rest until May, 1907, between eighty and ninety injections being given, nearly all in the pectoral region, a few in the tumour of the neck, none in the tonsil. Microscopical examination of the tissues removed from both tonsil and neck showed the growths to be round-celled sarcoma. The patient was very susceptible to the toxins; 2 mm. to 3 mm. was the highest dose given; this would produce a temperature of 103° F. to 106° F. Entire disappearance. Patient perfectly well at present, nearly three years.

(6) *Spindle-celled Sarcoma of the Sternum; Metastases in the Glands of the Neck* (diagnosis confirmed by microscopical examination made by Dr. James

¹ *Amer. Journ. Med. Sci.*, Philad., 1906, N.S., cxxxi, pp. 375-430.

Ewing, Professor of Pathology, Cornell University).—Mrs. G., aged 40. Treatment carried out under my direction by Dr. David John, of Yonkers; as high as 30 mm. given directly into the tumour; very severe reactions; showed little improvement until large doses were reached. Treatment continued for nearly six months; entire disappearance. Patient well at present, over three years.

(7) *Sarcoma of the Radius, Round-celled (giant); Spontaneous Fracture; Exploratory Operation with Curetting.*—Mrs. F., aged 26. Amputation advised by Dr. Frank Hartley and Dr. E. H. Pool, of the New York Hospital, in May, 1908; refused; mixed toxins for five weeks. Perfectly well at present, fifteen months afterward.

(8) *Sarcoma of the Ilium, very extensive, Inoperable.*—N. G., male, aged 13. Referred to me as an inoperable case by Dr. La Ferté, of Detroit, Michigan, in August, 1906. Intestinal trouble two years before; almost entire left ilium involved; toxins continued with intervals of rest for nearly a year; almost complete disappearance of the tumour. Patient in perfect health at present, three years later.

(9) *Spindle-celled Sarcoma of the Gluteal Region, Recurrent, Inoperable.*—F. L., female, aged 48. Apparent disappearance under three months' treatment; recurrence in pelvis the following year; inoperable; no further treatment.

(10) *Sarcoma of the Neck (Hodgkin's Disease).*—G. K., male, aged 20. Glands on both sides of neck involved, axilla, and groin; spleen markedly enlarged; one of the tumours of the neck removed, and microscopical examination by Dr. James Ewing showed it to be Hodgkin's disease. Entire disappearance under six weeks' treatment with the toxins; no further treatment; regained normal weight; recurred about a year later; could not be persuaded to resume treatment. Died six months later.

(11) *Small Round-celled Sarcoma of Tonsil with extensive Metastases on both sides of Neck.*—Mr. C., aged 42. Marked loss of weight; referred to me May 29, 1906. Prognosis seemed so hopeless that no microscopical examination was made; toxins pushed as high as 20 mm. in pectoral region; severe reaction; continued to lose in weight under treatment. After thirty-one injections, advised to go home for a rest; tumours had slowly decreased in size under the treatment. After leaving the hospital he took a patent medicine of some sort; growth continued to decrease in size, and four months later had entirely disappeared. In June, 1907, a year later, he had a recurrence in the neck; exploratory operation; microscopical examination showed it to be round-celled sarcoma. Resumption of the toxins advised, but refused. Died of the recurrence within a year.

(12) *Round-celled Sarcoma of the Groin.*—E. C. B., male, aged 20. Operation performed at Springfield, Mass.; microscopical examination by Dr. J. J. Butler, pathologist to the hospital, showed it to be small round-celled sarcoma; recurred shortly after operation; disappearance under the toxin

treatment; recurrence in two months; again disappeared under renewed treatment. Well at present, one year later.

(13) *Sarcoma of the Neck; Round-celled Lymphosarcoma*.—E. L., male, aged 38. Referred by Dr. Stillman, of San Francisco, California; microscopical examination made by Drs. Ophyls, of San Francisco, and Welch, of Johns Hopkins; partial removal by operation; remaining tumour disappeared under the toxins: injections given for six months. Patient well at present, nine months.

(14) *Sarcoma of the Breast and Axilla*.—Mrs. J. G., in December, 1905, first noticed a small tumour in the lower axillary region on the left side; this slowly increased in size until March 23, 1906, when it was removed by operation. The report of the pathologist of the West Pennsylvania Hospital showed it to be a lymphosarcoma. The entire tumour was not removed at the time of the operation. The toxins were begun by myself, and then continued by the family physician for about three months. The patient has remained in perfect health since, upwards of three years.

(15) *Sarcoma of the Right Deltoid Region (Spindle-celled); well six years*.—H. B. M., male, aged 56. Removed by Dr. W. A. Brooks, of Boston, September, 1902; the tumour originated in the deltoid region. Examination, February, 1903, showed a small mass in the lower extremity of the cicatrix, apparently a recurrence of the growth. Pathological report by Dr. Wright, of the Massachusetts General Hospital Laboratory: spindle-celled sarcoma. The toxins were given by myself for two to three weeks, and then continued by the family physician for about three months. The patient is well at present, six years later.

(16) *Large, Recurrent Spindle-celled Sarcoma of Cheek*.—F. Q., male, aged 63, in January, 1904, noticed swelling in the middle of the right cheek; removed one week later; recurred almost immediately, and grew very fast. March 2, 1904, the patient came under my care; at this time there was a hard mass in the central portion of the right cheek, about 2 in. in diameter and $\frac{1}{4}$ in. thick. An attempt was made to remove the growth, but this was impossible without sacrificing almost entire cheek. Pathological examination showed the tumour to be spindle-celled sarcoma. Immediately after the wound had closed the patient was put upon the combined X-ray and mixed toxin treatment, which was continued for about three months, at the end of which time the growth had disappeared and the cheek was apparently normal. August 15, 1909, patient in perfect condition.

At a recent meeting of the Medical Society of Greater New York I showed eight patients who had been treated for inoperable sarcoma with the toxins, and in three of the eight the limbs—leg in two cases, and arm in the other—had been saved from amputation. Seven cases well from two to fourteen years, and the eighth is well over one year.

In the discussion of these cases certain criticism was offered by a surgeon which is typical of the sort of criticism the method received in former days, but which has become less and less frequent in recent years. No criticism was made as to the accuracy of the diagnosis in these cases, nor of the fact that the tumours disappeared and the patients themselves were in perfect health. The patients were present as visible proof of the latter fact, but this was the line of reasoning: (1) The treatment, if of the value claimed, should—after fifteen years—have become generally accepted all over the world and universally adopted. (2) The critic had just returned from Europe, and stated that the treatment was not generally used or accepted there: *ergo*, it could not be of value. Furthermore, the critic had himself tried it in a certain number of cases many years ago, and had not obtained the same results as myself.

I will leave the answering of such arguments to others with greater love for disputation than myself. I will only call attention to one fact, apparent to anyone familiar with the history of medical discoveries, and that is that the relative value of such discoveries bear not the slightest relation to the rapidity of their acceptance by the medical profession. Numerous examples will doubtless occur to you, but few more striking than the one cited by Dr. Eccles in his admirable address on "Darwinism and Malaria," *New York Medical Record*, January 16, 1909.

In conclusion I cannot do better than quote from a recent and unpublished paper of mine: "It is natural that any new method of treatment of disease should stand a certain definite test before it can hope to secure recognition. When it comes to the consideration of a new method of treatment for malignant tumours, we must not wonder that a profession with memories overburdened with a thousand and one much-vaunted remedies that have been tried and failed takes little interest in any new method and shows less inclination to examine into its merits. Cold indifference is all it can expect, and rightly too, until it has something beside novelty to offer in its favour. Sixteen years ago, when I began to use the toxins for inoperable sarcoma, I did not expect the profession to adopt the method. I was perfectly willing to wait until its great objection of novelty had given way to time, and my own results had been duplicated and confirmed by other observers. No one could see the results I saw and lose faith in the method. To see poor hopeless sufferers in the last stages of inoperable sarcoma show signs of improvement, to watch their tumours steadily disappear, and finally see

them restored to life and health, was sufficient to keep up my enthusiasm. That only a few instead of the majority showed such brilliant results did not cause me to abandon the method, but only stimulated me to more earnest search for further improvements in the method."

The results within the last two years, due to Dr. Tracy's improved method of preparing the toxins, have been decidedly superior to those obtained before. They now comprise fifty-two personal successes and at least twice that number in the hands of other men, which, I think, should be sufficient to convince the majority of the profession that the treatment of inoperable sarcoma by certain bacterial toxins contains a principle of sufficient value to be entitled to more careful consideration than it has yet received, and if I have succeeded in impressing the Fellows of the Royal Society of Medicine with the correctness of this view, I shall feel more than repaid for my visit to England.

In closing, I wish to state that the results that I have reported would never have been possible without the help of others, and I desire to again express my deep sense of obligation to Dr. B. H. Buxton, Professor of Experimental Pathology at Cornell University (Medical Department), for early and continual help in the preparation of the toxins. Whatever efficiency they have is largely due to his skill and patient co-operation. I desire also to acknowledge a great debt to the late Dr. William T. Bull for most generous help and encouragement, especially in the earlier experiments with the living cultures. The large amount of clinical material could not have been obtained without his influence and co-operation. My thanks are also due to Dr. Martha Tracy and Dr. S. P. Beebe (of the Huntington Cancer Research Fund) for suggesting valuable improvements in the technique of preparing the toxins within the last two years.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said the Society was much more indebted to Dr. Coley for his very interesting address. They admired his perseverance in the investigation and the use of the method of treatment which he had described. It had been said that there was not sufficient scientific basis for the treatment, but Dr. Coley had shown that he arrived at his method by carefully considered investigations, which it might be hoped that he would continue successfully to prosecute. Moreover, in the treatment of disease they had all used with advantage remedies of which they had been unable to give a scientific explanation, and had relied on clinical results to justify them in such

treatment. They might hope, therefore, that increased experience and careful records of Dr. Coley's method would enable them to judge more accurately of the appropriate dose of the fluid and the frequency of administration for each case; and in the meantime it seemed to him that what Dr. Coley told them was sufficient to encourage them to try his method in a disease such as sarcoma, many cases of which were so hopeless.

Mr. BUTLIN said: I came down expecting a discussion on the address, and nothing would have given me greater pleasure than to take part in it. Although Dr. Coley and I have never met, we are old friends, because we have corresponded regularly. He has been good enough to send me copies of his papers from time to time. He has not only done that, but he has also sent me material for use, the mixed bacilli of erysipelas and prodigiosus, and I have used it at different times. Of course, I cannot talk about his address, because that would open a discussion, and Dr. Coley would not have a proper opportunity for replying. But I should like to tell this assembly what I know of this treatment of Dr. Coley's. During the last twenty-five years I have been largely consulted on malignant disease, and I have seen many different kinds of so-called "cancer cures." These cures have all gone through the same kind of course. In the first stage the cure was to cure cancer; in the second stage, the cure was not to cure cancer, but it was to retard its progress and prevent recurrence; and the last and third stage of a cancer cure was the stage of oblivion. With the exception of Dr. Coley's method, scarce a single one has survived. Dr. Coley's fluid was introduced sixteen years ago or more. In the first instance, it was hoped that it would cure not only sarcoma but carcinoma. Then it was found to have no effect on carcinoma, but it was still hoped it would cure sarcoma. If not, it was hoped it would prevent recurrence and relieve pain. Later, it had nearly fallen into the stage of oblivion, for, in addition to the natural distrust which our profession have of any kind of new cure for cancer, a sub-committee consisting of some of the best surgeons in America, appointed by one of the great American medical societies, inquired into Dr. Coley's method, and came to the conclusion that the fluid was useless against malignant disease. That was enough to damp the ardour of anyone but Dr. Coley. He, however, had such full confidence in his treatment that he continued to use it and to recommend it, until he has succeeded in convincing not only America but this country that it deserves the closest attention, and that an extended trial should be given it. The further history of cases of sarcoma of the bones of the lower extremity, alone, is sufficient to convince me that Coley's fluid is a very potent medicine. I have, until now, looked on sarcoma of the femur (unless it be giant-celled) as an invariably fatal disease. But a number of cases of sarcoma of the femur are now reported as living some years after the treatment by Coley's fluid, either with or without operation. The difficulties in the way of adopting Coley's treatment are: The preparation of the material, the discomfort and malaise produced by the injections, and the rare occurrence of cases of sarcoma. On the first and second of these difficulties Dr. Coley has furnished implicit instructions, and these should be carefully studied by those who

would practise his method. On the third of them, I would say that sarcoma is rare in the practice of each individual hospital surgeon; so rare in private practice that, during the last seven years in which I have been consulted on malignant disease, I doubt if I have seen one case of sarcoma in each year. I have very great pleasure in proposing a hearty vote of thanks to Dr. Coley for his address, and for the devotion to his art which he has shown in travelling all this distance (from America) to deliver it. He has already made converts in this country to his views by his printed papers. His visit will strengthen the faith of those who mean to try his fluid. I only wish America could spare him to us for six months or a year, that he might himself superintend the treatment of a series of cases.

Sir A. E. WRIGHT, in seconding the vote of thanks, said that it had been inspiring to hear from Dr. Coley's own lips an account of the wonderful results he had achieved in the treatment of sarcoma; and he felt it a privilege to express on the part of the Society its debt of gratitude to Dr. Coley for his address. He was convinced that, whereas in the case of this method of treatment the results which have been achieved were known only indirectly on the testimony of witnesses, there was nothing which was to those who wished to arrive at a correct judgment so helpful as the actual seeing and hearing of the author. He felt sure that Dr. Coley had inspired everyone in his audience with the conviction of his veracity and with a conviction of the reality of his cures; and in listening to him everyone must have appreciated how severe must have been the strain undergone in contending with a reputedly hopeless disease, in employing a method which involves the infliction of severe local and constitutional suffering and possible risk to life, and in carrying on such work in the face of chilling scepticism. Dr. Coley was, he thought, entitled to receive their warmest congratulations and their tribute of admiration for the pluck and grit and doggedness which he had displayed in carrying his work through to results such as he had just recounted. There was, however, another aspect of Dr. Coley's work which ought, he thought, to be adverted to. Dr. Coley's method apparently rested on a purely empirical basis, and it was applied without any kind of scientific control. So distant, in fact, were the relations between this method and scientific thought that Dr. Coley's addresses had left his hearers in doubt as to whether he administers his bacterial filtrates with intent to produce a direct toxic and necrotic effect selectively upon the neoplastic cells, or whether he administers them as vaccines on the assumption that we have in sarcoma a concomitant streptococcal infection, or whether he employs them after the manner of a quack medicine without any thought of their mode of action. He confessed that he would have been grateful to have learned from Dr. Coley of some method of standardizing bacterial filtrates which are employed; he would have been glad to have learned why filtrates of prodigiousus and no others are added to his streptococcus filtrates; and, above all others, he would have been glad to have learned what changes are produced in the blood by successful inoculation in order that these might be employed in controlling and guiding the treatment. In all these hopes he had been disappointed. He

thought he could detect the same note of disappointment in Mr. Butlin's suggestion that steps should be taken to persuade America to lend Dr. Coley to England for a twelvemonth to take over the direction of his treatment in this country. Mr. Butlin was perhaps reflecting that in the absence of Dr. Coley we have no means of conducting his treatment aright. There here stood revealed, he took it, a great lacuna in Dr. Coley's work. Until that lacuna had been filled up by laboratory work done in conjunction with Dr. Coley's treatment, he was afraid it was impossible to feel any assurance that the results which had been achieved by Dr. Coley would be again realized here.

Dr. COLEY, in replying to the vote of thanks, said that he desired to express his deep appreciation of the interest shown and the gracious reception given his address by the members of the Society. He hoped the Society would permit him to say a word in reply to the proposer and seconder of the vote of thanks. In response to the remarks of Mr. Butlin, regarding the adverse report upon the value of the mixed toxins made by the Committee of the New York Surgical Society twelve years ago, he would say that that Committee was composed of three men. One of these, Dr. A. J. McCosh, had since been convinced of the value of the toxins, and had sent Dr. Coley a number of cases for treatment with the toxins during the last few years. The second member of that Committee was Dr. A. G. Gerster, and he had been convinced of the value of the toxins so thoroughly that he employs them at the Mount Sinai Hospital, not only in inoperable cases, but uses them after primary operations as a prophylactic against recurrence. Dr. Coley stated that, some four years ago, Dr. Gerster had sent him a patient with primary round-celled sarcoma of the tonsil, with extensive metastases in the neck. A partial operation had been performed, but the growth was too extensive for complete removal. The diagnosis had been confirmed by microscopical examination made at two laboratories. One large hospital had given him but a few weeks to live. The patient was admitted to the General Memorial Hospital in October, 1905, and under seven weeks' treatment with the mixed toxins the tumour entirely disappeared. Dr. Coley then read two letters from Dr. Gerster, one written directly after the tumours had disappeared, and the other after he had examined the patient and found him well, three years later. The letters were as follows :—

Copy, Letter No. 1.]

NEW YORK, December 1, 1905.

MY DEAR DR. COLEY,—It is my agreeable duty to congratulate you most sincerely on the brilliant achievement of the treatment you have given to our patient, Miller. Most certainly I have never seen anything like it. The tonsil looks now like a normal organ, and the large glandular and periglandular swelling of the neck has entirely disappeared. I hope it may turn out to be a permanent result. Thanking you for the interest taken in this seemingly hopeless case, I remain gratefully yours,

ARPAD G. GERSTER.

Letter No. 2.]

NEW YORK, June 17, 1908.

MY DEAR DR. COLEY.—Thank you for your note informing me about the brilliant and lasting result in the case of Harry Miller. It is the most gratifying case within my experience. May you have many more such! Truly yours,

A. G. GERSTER.

Dr. Coley further stated that he had shown this patient before the Society of Greater New York, in February, 1909, in perfect health, with no trace of the tumour either in the tonsil or neck. He added that the patient remained well at present. Dr. Coley stated that instead of in the beginning advising the mixed toxins as a method of treatment for cancer in general, he had begun by strictly limiting it to cases of inoperable sarcoma, and only after long experience had he felt justified on basis of the results obtained in extending its field of usefulness as a prophylactic after operation, and before operation to the limited group of cases where operation meant the sacrifice of a limb. With reference to the remarks of Sir Almroth Wright, Dr. Coley stated that he must take exception to the method being classed as purely empirical and in the nature of a quack remedy, without laboratory basis. He had tried to show in his paper that the method was an absolutely scientific one: (1) Because it was founded upon a long array of demonstrable clinical facts—namely, a large series of cases of inoperable sarcoma that had disappeared under attacks of accidental erysipelas, and the patients had remained permanently cured. (2) The method rested upon the further fact that in a large number of cases of inoperable sarcomas treated with the mixed toxins of erysipelas and *Bacillus prodigiosus* the tumours had disappeared, and a goodly proportion of these cases had remained well from three to fifteen years, the diagnosis having been verified clinically and microscopically by the most competent men in surgery and pathology. (3) The addition of the *Bacillus prodigiosus* was not an empirical thing at all, but rested upon laboratory investigations of Roger, who found that the *Bacillus prodigiosus* grown together with the streptococcus of erysipelas greatly increased the virulence of the latter. It was this fact that led Dr. Coley to try the addition of the *Bacillus prodigiosus* to the erysipelas toxins. (4) The method during the last two years had been submitted to actual laboratory tests, and the investigations of Dr. Martha Tracy and Dr. S. P. Beebe, of the Huntington Cancer Research Fund, at the Loomis Laboratory, Cornell University Medical School, had demonstrated that multiple sarcomas in dogs disappear under injections of the mixed toxins, and, still more important, under the injections of the *Bacillus prodigiosus* alone.

Surgical Section.

October 12, 1909.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Prognosis in certain Inflammatory Diseases of the Lungs and Pleura commonly treated Surgically.

It is unusual for a new President to introduce a subject or start a discussion, and so some explanation, if not apology, may be expected. When elected to this honourable position (and I take this earliest opportunity of saying how much I feel its dignity and am oppressed by its responsibilities), the first news was that there were no papers for the next session. It is a difficulty which all secretaries recognize as incidental to our method of accepting papers, as it is natural that authors should not like their efforts to lie dormant for three months. My paper has been submitted to no censorship. When it has been read you will recognize its many defects.

I have chosen a subject of great importance: the *prognosis* in some of those common inflammatory diseases of the chest which belong at least as much to the physician as to the surgeon. They cannot be called surgical diseases, for, indeed, in one class the physician often does all the surgery that is required. And, moreover, in all, when the surgeon has done his part, the "case," if it remains a case at all, is essentially medical. That is why the subject is so difficult to work up, and it soon became evident that a much longer time was required to do justice to it than the two holiday months which were at my disposal.

It was impossible to get at the notes of hospital cases, and, after all, it is not easy to follow them up; so it was necessary to fall back upon private notebooks and, though they supply records of a considerable number of the rarer diseases, the more common and therefore the more valuable are not so largely represented as I could have wished.

I cannot, therefore, produce long tables of statistics, but I would not do it if it were possible, for they would be of little or no value. Suppose, for instance, one were to tabulate the cases of acute empyema without noting the age (I do not mean whether the patient is an infant or an adult, but whether a young or old adult), or without specifying the organism which is the *causa causans*, or the length of time that the case has been "watched" before an operation is done, or the nature of the operation performed, such tables would be worse than useless. It is, unfortunately, true that acute empyemas are frequently recognized days and days before the surgeon is called in, till the patient becomes more and more poisoned and the lung more and more compressed, and then the prognosis is principally concerned with the next ensuing few hours or days; whereas if promptly dealt with the immediate risk would have been small, and the question would have turned on the chances of complete or partial recovery.

If the criticism is made that only a series of cases supposed to be illustrative is produced, it may be pointed out that from such a source a far more important kind of information may be obtained (provided only that the series is large enough) than that which is derived by striking an average from a very large number. The object of discussing the question of prognosis is to help us in prophesying the future of the particular persons who seek our advice, and it is very unlikely that they will conform to the average. It is, of course, quite possible that not one of a long series exactly represents the average. A correct prognosis is more likely to be reached by comparison with the most similar individuals of the series rather than by fixing the attention on the *type*.

The first and simplest condition is that of *serous effusion into the pleura associated with acute pleurisy*, and I must begin with the lame admission that I have hardly any records to produce. I suppose we shall all agree that patients who have had one attack of acute pleurisy with effusion and have been tapped once or twice, or not tapped at all, usually leave the court without a stain upon their characters. They are admissible by insurance companies at normal rates, and the only record that remains (and that not always) is a slight departure from the normal physical signs, and perhaps a diminution of the mobility of the side that has been affected. The acute onset usually stamps their simple character. It is different when the onset is insidious. It has sometimes been suggested that most of the insidious cases are tuberculous. But it is no more true that they are all of this nature than

it is that all the tuberculous effusions are insidious in their onset, as the following two cases will show :—

Miss M., aged 22, whose illness began insidiously three weeks before she sought advice on account of a cough and cold, had her left chest full, and it was tapped out October 29, 1892, seventeen years ago. She "never looked back," so her stepfather, who is a doctor, writes. She married, and has two fine boys, aged 12 and 9, and has had no lung trouble since. (Dr. J. Waugh.)

Or this :—

A strong and apparently robust member of our own profession, aged 58, woke one morning with pain at the right base, which he thought was muscular rheumatism. He went on with his work for two days, and in a fortnight's time his chest was full (5 pints) of clear fluid, his temperature varying from 99° to 100° F.; pulse, 100; respiration, 22'40. No tubercle bacilli were found in the first fluid drawn off; but he died within a few weeks (after a second tapping) of acute tuberculosis. (Sir T. Lauder Brunton and Dr. Segundo.)

Still, I should expect an acute case to be caused by some septic organism, and should be suspicious that a chronic one would turn out to be tuberculous even if no tubercle bacilli were found microscopically or by inoculation, with the mental reservation, however, that it might depend on a new growth. But how much caution is needed in dealing with chronic cases!

I have seen a strong man, aged about 40, who simply complained of shortness of breath. His right chest was full of fluid, and that appeared to be all. In a few days, however, the left chest filled with bloody fluid. Then he quickly got thrombosis and gangrene in his limbs, and died of general carcinoma of both pleuræ. (Dr. Symes Thompson.)

One patient, a middle-aged man, I saw in 1893, had a large right serous effusion. There were no doubt other suspicious symptoms and signs in the chest and abdomen, so we were not surprised when he died a year later of rupture of an aneurism of the thoracic aorta. (Sir R. Douglas Powell and Dr. Nix.)

I might mention others where the effusion has been connected with trouble beneath the diaphragm, such as hepatic abscess, appendicitis, gastric ulcer, hydatids, and so forth; but these hardly concern the present discussion, except inasmuch as they raise the following question :—

What is the outlook if a normal pleura is opened and drained? It has often been stated that an abscess of the liver may be safely opened through a normal pleura without any special precautions for closing the opening. This is not my experience. I have seen it followed by fatal results in septic cases or by very slow healing in amœbic ones. That is why I avoid injuring the pleura, if possible, and carefully close the wound if it has been made.

I have a patient who in 1893 had that rare condition, hydropneumothorax, caused by a hydatid of the liver. The hydatids were removed through the incision. Various reasons have prevented any attempt to reduce the extensive cavity by means of a thoracoplasty, and he still wears a tube. So he has gone on for sixteen years, and has reached the age of 54; to some extent handicapped, of course, but enjoying life and actively engaged in business and amusements. He nearly died some years ago of acute pneumonia of the sound lung, but he has no sign of amyloid disease. (Dr. J. Mitchell Bruce.)

Are we justified, then, in incising and draining serous effusions that reaccumulate? To this different answers will be supplied. For myself, I am so afraid of leaving a permanently open pleura with collapsed lung that I advise repeated tapping, even up to seventy times seven, tedious though the process is. The following case is an example of its employment:—

Mr. S., aged 52, whose illness began insidiously, when influenza was prevalent, in January, 1906, had effusion at the right base without signs of pneumonia; $2\frac{1}{2}$ pints of clear fluid were drawn off containing "a due proportion of leucocytes, a few red discs, no tubercle bacilli, and no pyococci." The temperature was 90° to 101° F.; pulse 90 to 100. He was tapped again on March 1, March 10, March 14, and altogether sixteen times. Fluid also formed on the left side and was drawn off twice. The treatment was completed in seven months. He is now in good health, goes to town for business and Switzerland for pleasure, drives a motor for excitement, and plays golf for repose. He gets a certain amount of catarrh at times, but no tubercle bacilli have ever been found in the fluid. (Dr. Blomfield.)

A more puzzling case is the following:—

Mrs. G., a delicate lady, aged 58, who for thirty-five years had been liable to hæmatemesis, developed double pneumonia in January of this year, with moderately high temperature and rapid pulse. She was tapped four times on the left side, the fluid being slightly stained with blood. There was very rapid reaccumulation on every occasion except the last, on March 25, since which she has lost all her chest symptoms, though there has been another attack of hæmatemesis. (Sir T. Lauder Brunton and Dr. Gill.)

I interpose here the mention of a mistake in prognosis, or perhaps I ought to say diagnosis, which I made three years ago:—

C., a man, aged 52, who had had a so-called "bilious attack" with jaundice and blood in the motions twenty years before, and no other similar attack, began to lose flesh in the summer of 1905, and had a sort of biliary colic in December, after feeling ill for three days. There was no jaundice. The temperature was erratic, varying from normal to 103° F. The liver, or what felt like the liver, was large and hard, smooth and fixed. These symptoms were followed by copious hæmaturia and melæna and the accumulation of fluid

in the right pleura. When I saw him on February 13, 1906, he was much emaciated, with a flabby pulse of 120 to 130. I drew off about a pint of slightly turbid, pale, opalescent fluid, and said that I thought it was probably a case of malignant disease about the region of the head of the pancreas, and offered to explore the abdomen if desired. Not long afterwards, however, he consulted Mr. Mayo Robson, who has reported the case in the Hunterian Lecture.¹ It appears that in the interval a change had occurred—for there was a large collection of pus and gas below the diaphragm—and that the cause of all the trouble was a duodenal ulcer, for which Mr. Robson did a gastro-enterostomy on a subsequent occasion, which was followed by complete recovery, and the patient is now in excellent health. What the relation of the hæmaturia was to the original disease, if any, still remains unexplained. (Sir T. Lauder Brunton and Dr. Griffin.)

No doubt this will call up in the minds of many problematical cases, some of which have been explained by subsequent events, while others have always remained mysteries—such, indeed, as thoroughly warrant the uncourageous note: *prognosis doubtful*.

An effusion which is undoubtedly tuberculous need not necessarily involve a very grave prognosis, one tapping being sometimes enough to effect a cure.

Such was the case of B. S., aged 47, whose illness began in 1895 with right pleurisy with a rigor, after he had been nursing a brother who had died of acute laryngeal tuberculosis. He had a considerable serous effusion on the right side which did not reaccumulate after a single tapping, and he followed his profession in comparatively good health for a long time, and survived the tapping thirteen years. (Dr. J. Mitchell Bruce.)

Passing now to the subject of *empyema*, I must first offer some general remarks about the *acute empyema of infancy*. In 1886 I gave my statistics when I was surgeon to the North-Eastern Hospital for Children, and the average time for closure was six weeks. But this did not represent the usual course of things: three or four weeks was a common time for an acute empyema, and ten days or a fortnight was not rare, while a good number were cured by a single aspiration. The average was obtained by including the chronic cases. I do not gather that much better results are obtained now, although one might imagine that each successive writer on the subject had discovered some new panacea. A free opening, good drainage, in the recumbent as well as the erect posture, the complete removal of all fibrinous masses, and the exercise of a wise judgment in removing the drainage-tube are essential to success. The precise spot to half an inch at which the opening is

¹ *Brit. Med. Journ.*, 1907, i, p. 248.

made, the introduction or not of the gloved finger, and the presence or absence of an antiseptic material in the dressings are matters of less vital importance.

I have placed these together without forgetting that many different organisms are accountable for them, and that probably a careful comparison of these might lead to the formation of a more accurate prognosis. Because the soft chest-walls of children and the elasticity of all their structures, and perhaps their more active vital powers, seem to favour the rapid closure of an empyema. It gives the opportunity also to produce a few records which I have traced from youth to maturity:—

I. R., aged 4, was brought to me in June, 1896, with localized empyema imperfectly opened in the axilla, which had followed double pneumonia. I enlarged the opening and she rapidly recovered, and has been well ever since—i.e., thirteen years. (Dr. Warren.)

H. H., aged 9, had a right empyema following pneumonia, associated with measles, which opened posteriorly on April 15, 1893. There was gradual subsidence of the fever, and it was healed by the end of May. Now (sixteen years after) he is well and strong, and has had no chest trouble since. It is curious that his father, who was a strong man and came of a sound stock, had afterwards an effusion in his left pleura, which was tapped, and, though he made a good recovery, he died some years later of pulmonary tuberculosis. (Dr. Rushworth.)

P. M., aged 11, in 1890 had an empyema following some septic poisoning that affected several boys at the school. It was opened (not by me) in the sixth interspace in the axilla; I think it was pointing there. It was said that he had had endocarditis, and he had a very localized apex murmur. When I saw him in 1892 the wound had long been closed. He went to Cambridge, where he rowed and played lawn-tennis well. He is now 6 ft. 4 in., and is professor at an engineering college in India. He stands the climate well, and he had no further chest trouble—i.e., during the last nineteen years. His strenuous life has occasionally resulted in a certain amount of neurasthenia. (Dr. Newman.)

Master S., aged 10, had a right general empyema, which was opened posteriorly on March 31, 1886, and healed in three weeks. He has had no illness since—i.e., twenty-three years. He went to Oxford, and is now a curate who is said to have a good preaching voice. (Dr. Burton.)

Such cases are, I believe, common—almost typical. I wish the medical officers of insurance companies would tell us something about them. They must often apply for life insurance, and one would like to know whether any increased rate is imposed upon them. I should myself not advise it in spite of such cases as the two following:—

Master R., aged 14, a thin, delicate-looking public-school boy, with a long phthinoïd chest, had a large right empyema, which I opened posteriorly on April 28, 1886. There was an aseptic course, and the wound closed on June 9. He recovered and put on much flesh, but died twenty-one years later of phthisis after severe hæmoptysis. (Sir R. Douglas Powell and Dr. Drew.)

Miss C., who is now aged 28, had a left empyema when she was aged 2. It was imperfectly opened in the axilla, and she was brought to me two years later, in 1885, at which time there was much clubbing and great deformity of the chest. I did a thoracoplasty, and she much improved, and eleven years later was well grown; the clubbing had almost gone, but the sinus was not healed, and no doubt communicated with the lung. In 1889 she had had signs of pulmonary osteo-arthritis, which had also disappeared. In 1906 I removed some portions of rib, opened up the track, and found a sequestrum in it. The wound is now healed, and though there is very great deformity of the chest, and though she is a delicate woman, she leads an active and useful life in India. (Sir T. Barlow and Dr. Donaldson.)

I am afraid the particular answers desired are not to be obtained from insurance companies. It does not help us to know the average age of the patients insured (say in the Scottish Widows' Fund) who die of pleurisy and empyema, and it does not seem possible to obtain information as to the results from the point of view of the insurance companies of accepting proposals from those who have suffered from hydrothorax or empyema in childhood. As far as I can gather, medical officers do not often have such proposals made to them, and, if they do, are very shy of entertaining them. Dr. Reginald Thompson, a man of very wide experience, writes:—

"I will not pass doubtful lives. I have had very few cases of pleurisy with effusion (and paracentesis), and still fewer of empyema, to give an opinion upon. I should reject all cases of empyema and look askance upon cases which have been tapped for serous effusion; but I might take the latter cases at ordinary rates provided all other conditions are favourable—viz., family history, personal history, and weight. My colleague Kidd thinks that all such cases are connected with tubercle. That is not my opinion."

Turning now to *acute empyemas in adults following acute pneumonia or pleurisy*, the immediate prognosis as regards life depends upon the extent of the preceding disease and the amount of septic poisoning. The latter is indicated by the character of the pus; if this is sweet and only contains pneumococci, the patients do not necessarily present what may be called an obviously septic appearance. If they do, and the pus is odourless, it probably contains streptococci or staphylococci. If it stinks, there has probably been a rupture of a pulmonary or sub-diaphragmatic abscess into the pleura. The pneumococcus cases, if

treated carefully antiseptically, I think do the best, especially if the lung is still somewhat solid from the pneumonia and the cavity is localized; but I have often seen (especially in hospital) a secondary pyrexia associated with a secondary septic infection. It is remarkable how well many of the stinking cases do. Some of the very worst septic ones may be snatched from death by continuous or repeated saline infusions or transfusions.

Undoubtedly the great majority of these cases survive the operation, and I am not concerned with the small number who die within a few hours or days of it. An accurate prognosis as to the date of closure is impossible. Speaking generally, it may be said that the least favourable cases are those in which the cavity is large, or the lung is much collapsed, or the chest-walls very rigid, and those in which there is a very free communication with a bronchus. My own opinion is that those which are opened laterally are more likely to be slow in healing, or to require a second opening, than those which are incised posteriorly. So I think that the prognosis is to some extent governed by the surgery.

One would naturally expect that old people would do worse than young ones, but some heal remarkably quickly, e.g. :—

Mrs. T., aged 69, had pneumonia followed by a localized left empyema opened May, 1900. It healed in a month. She was seen in July, 1901, and was then quite well, and was certainly well for "a year or two after 1903," since when nothing has been heard of her. That would be five years, and bring the patient's age to 74. She is probably still alive. (Mr. S. N. Bruce.)

J. R., aged 75, had influenza and pneumonia followed by a large empyema opened January 6, 1909. It healed in a few weeks, and he still keeps quite well, though not quite so strong as before his illness. (Sir T. Lauder Brunton.)

Of the cases that heal, the great majority remain well or have very slight inconveniences left behind. A small number have recurrences and some develop bronchiectasis. Of the last class I have only notes of one example, a man who died when aged 55, twenty-five years after his empyema had burst spontaneously. I will now give some examples of the other two classes, and then deal with those that do not heal :—

Mr. M., aged 55, had influenza and right pneumonia in January, 1907. An empyema was opened on February 2; it healed rapidly, and he remains well. (Dr. Bland.)

V. I., aged about 45, had pleurisy in June, 1906, and a localized empyema, which was opened in the right axilla in June, 1906. There was also some friction at the left base. He rapidly recovered and remains quite well. (Dr. Krott.)

Mr. E., aged 49, had right pneumonia. Stinking pus was removed by aspiration on the sixteenth day. Free incision was made posteriorly on the eighteenth day in February, 1904. It healed in eight weeks. He is now quite well, but some friction at the right base was heard in May, 1905, after an attack of influenza. Nothing, however, came of it.

Mr. H., who was shot when serving with Montmorency's Scouts, had absolutely recovered from a very bad and extensive septic empyema, for which I had treated him nine or ten years before—viz., 1890. He became a fine, strong, active man, who took no precautions with regard to his health. (Dr. Kidd.)

Mr. A., aged 47, had a left empyema of small size. The pus was stinking. It was opened on November 26, 1900. It healed rapidly, and he has had no further trouble. (Dr. Scott.)

G. P., aged 25, was shipwrecked in December, 1899, on his way to the Boer War. Then he had pneumonia and empyema. There was much delay in opening the latter, which was done in June, 1900. It healed in August of the same year, and he is now quite well. (Dr. J. K. Fowler.)

Mr. T., aged 38, broke his clavicle while hunting in December, 1898. After three weeks he had "influenza" and pleurisy. This was tapped abroad. Pus was expectorated in March, 1899, but there was great delay in opening the empyema. This was done in June, 1899, and, though it was very extensive and there was a bronchial fistula, it healed in seven weeks, and he has remained well ever since. He now hunts five days a week in the season. (Dr. Bookham.)

Mr. P., aged 48, had left apical empyema following influenza. It was opened in the first interspace in May, 1893: it quickly healed. He had remarkably good health afterwards, and there has been no further trouble with this side; but after an attack of influenza two years ago a patch of dullness appeared on the opposite side, and he kept his bed for fourteen weeks. This has left him less active than before. (Dr. Parrott.)

G. F., aged 24, had a large right empyema following pneumonia caused by a chill. It was opened October, 1892. It healed in six weeks. He is quite well except that he cannot take active exercise, like cricket or rowing, because he gets out of breath. (Dr. Peter Cooper.)

Mrs. D., aged 30, had left pneumonia and empyema in March, 1892. It healed in a few weeks. She is now well except for attacks of asthma and bronchitis, to which she has always been subject, and that her breast was removed for scirrhus five years ago. (Dr. Gross.)

Mr. S., aged 39, a master at a public school, had a bad left empyema following pneumonia, which was operated on in June, 1891—i.e., eighteen and a half years ago—and he reports that his health is precarious and that he is liable to bronchial colds, but no actual bronchitis or pneumonia. (Dr. Scott.)

Miss W., aged about 30, a hospital nurse, had a septic throat while nursing a patient with a similar condition in December, 1890. This was followed by

double empyema, possibly pericarditis and acute suppuration of one hip. Both pleuræ and the hip-joint were opened and healed. The recovery as regards the chest appeared to be perfect, and she remains well in that respect; she has, of course, a stiff hip, and has developed osteo-arthritis. (Mr. Howard Barrett.)

Mr. A. was aged 54 in 1887, and had then suffered for thirteen years from locomotor ataxy. He then developed a left empyema of moderate size containing stinking pus, which had ruptured into the lung three weeks before I opened it. Shortly after the operation he nearly died of septicæmia, in fact his life was despaired of; but he unexpectedly recovered completely as far as his chest is concerned, and is still alive after twenty-two years, active in mind, but feeble in body. He is very deaf and much emaciated, no doubt as a result of the morphia which he takes freely on account of the constantly recurring lightning pains. (Mr. Farnell.)

Amongst this list of thirteen cases there are nine who are alive and well, after more than nine years, of which six have survived the original illness more than sixteen, and one as many as twenty-two years. It is one that could be added to almost indefinitely, but it is a convenient place to turn to another class—namely, those where, as in the last case, *a fistula exists*—and to ask how far this influences the prognosis.

Although it is an undoubted fact that a small proportion of *empyemata which burst into a bronchus* recover completely in the course of a few days or weeks, and that such a cure may be permanent (I have records of one patient who remains well after seventeen years, Dr. W. Murray), it may be stated that the majority of them require an external incision; but in most of these the fistula quickly closes and healing occurs as if there were no such complication. It would be remarkable that such a result may be obtained, even though the external opening has been long delayed, if we did not remember what occurs in the treatment of pulmonary abscesses:—

J. E. H., aged 40, began to expectorate pus in 1885. When I saw him in 1893 (eight years later) he was bringing up a teacupful a day; he had then copious albuminuria and much clubbing. The opening of a large left empyema was followed by rapid closure and recovery of health. I saw him two years later and have heard of him since. He became very active and lost his clubbing, and there was much less albumin, though it did not disappear altogether. He apparently passed a stone from the right kidney afterwards. I think he is still alive, but I cannot trace him. (Dr. Harris.)

It is probable that some of the cases in which a permanent fistula remains are complicated with bronchiectasis. These may not only survive but retain good health for years. Such a case is that of—

J. K., aged 29, whose illness began in 1888 with congestion of the lungs and pleurisy, followed by mucous and later by bloody and offensive expectora-

tion. I opened a localized left empyema containing air four years afterwards, in 1892, which was followed by complete restoration of health. He lives in Australia, but I see him from time to time. He is in active business and weighs 13 st., as compared with 9 st. 6 lb. in 1890; the fistula is still open; the tube was 2 in. long when I last saw him, in 1907; the discharge, which is mucous, would cover the thumbnail on a bad day; he washes the tube daily, and renews it every three months. (Dr. C. T. Williams and Dr. Brogden.)

I have seen several cases like this and have attempted (unsuccessfully, however) to close the fistula. But some of them succumb to complications after improving for a time; in these one should be on the look-out for tubercle. Such a case is the following:—

L. O., aged 18, of tuberculous stock, had a serous effusion on the left side in 1893, and an empyema was opened in June of the same year. In November an abscess in the lung was evacuated through the original wound; she got much better, in fact she enjoyed fair health for ten years, but she could not do without a tube or a solid plug. She died in 1903, on the sixth day, of an attack of acute pneumonia. (Dr. Colegate.)

I will next consider a small class of cases *which heal readily and rapidly and remain well for longer or shorter periods—months, or perhaps years—and then the trouble recurs*, and perhaps not only once, but on several occasions. No doubt a spicule of dead bone is sometimes the cause, or a ligature, or perhaps a minute piece of necrosed tissue, but one often fails to discover such a foreign body. One recurrence need cause but little anxiety, but a second or third should make one speak doubtfully.

I have no record of an early patient of mine, K. G., who had repeated attacks, lasting over many years, and who at last died of an acute fever out of reach of me, and convinced himself that one of his customary operations would have saved him. But these are examples. (Dr. J. Mitchell Bruce.)

T. S. C., aged 57; had pleuro-pneumonia April 16, 1907, and stinking expectoration May 13. The empyema was opened May 23; the contents were very offensive. Recovery was rapid, and he had good health for two years—viz., till July, 1909—when the empyema refilled with stinking pus, and a similar material was expectorated; no foreign body was discovered. It was not quite healed by October 1. (Mr. Winstanley.)

Miss E. J., aged 14, had left pleurisy followed by empyema in February, 1896, which healed in eight weeks and remained well till October, 1897. Then for the first time she had offensive expectoration, and after a fortnight the empyema was reopened and quickly healed, and remained closed for a year, when it refilled and healed in eight weeks. The process was repeated in December, 1899, and in March a small piece of bone came away; but it filled again in 1900 and 1901. This time the sinus remained open for three years,

her health all this time being perfectly good; soon after, however, she had "influenza" and a large abscess in the right thigh, and she died exhausted. I only saw her once—viz., in 1900. (Dr. Simpson.)

Sometimes recurrence appears to follow exposure.

One such case occurred in the person of a pioneer in the Congo, who neglected the advice that Sir R. Douglas Powell and I gave him not to return to his duties so soon, and he fell a victim to the morphia craze from having been allowed the indulgence by a ship's surgeon. He is not the only one amongst my cases in whom a favourable prognosis has been thus falsified.

Sometimes the *healing process comes to a standstill* because the drainage is imperfect or because the cavity is too large. The latter condition may be anticipated where no adhesions have formed, and especially where none exist between the base of the lung and the diaphragm, or the inner surface and the mediastinum. These most unpromising cases result from the rupture of a pulmonary infarct, a liver abscess, or something of that sort into a previously healthy pleura. The immediate outlook here is serious, as, if not quickly opened, the patient may die within a day or two. The ultimate prognosis is also grave, as the cavity is sometimes so large that it cannot be closed by the most extensive *thoracoplasty*. Should the *thoracoplasty* succeed the prospect is, I believe, as good as that of an empyema which has closed by natural processes. It should be observed that extensive thoracoplasties are not without their dangers, such as shock, septic absorption, and cerebral abscess.

It will be interesting to inquire as to the future of the *cases which remain permanently open*.

One of my earliest was that of a strong young labourer whose empyema followed an accident, and was opened, after the imperfect methods of those days, in 1876. He was an obstinate man and would never submit to a second operation, in spite of my urgent entreaties, but continued to drain his cavity through a No. 13 gum-elastic catheter, which usually stank horribly. For eight years he remained in excellent health, in spite of contracting syphilis. Then amyloid changes began, and he very gradually went downhill; but he was certainly alive in 1892, sixteen years after his first illness.

Another hospital patient, aged about 25, whose empyema was opened by Mr. Barker in 1883, and who was subsequently operated on by Mr. Marshall, Mr. Beck and myself, lived till 1890, seven years, in spite of the fact that he was of drunken habits, and when he lost a drainage tube simply inserted another; so that seven were found in his pleura at the autopsy.

Mr. C., aged 25, who, as far as I know, is still alive, told a curious tale. He said he was shot through the chest while looking down his pistol, which had

been surreptitiously loaded by his Indian servant in October, 1895. Pericarditis and pleurisy followed, and a left empyema resulted, which was opened in the axilla; and there was a large cavity discharging about 2 oz. of pus daily, and an unexpanded lung, when I first saw him nine months afterwards. I made him a posterior opening, with the result that the discharge practically stopped, but the lateral incision did not close completely; a minute track into his practically unaltered pleura remained. In fact, he was in the condition of a man with pneumothorax communicating by a pin-point opening with the outer air, which he covered with a piece of plaster. With this state of things he kept in good health, and there was no discharge unless he took excessive exercise, such as running to catch a train, a few hours after which a few ounces of serum would escape; but often nothing would appear for months at a time. Unfortunately he developed the morphia habit, and was very thin when I last saw him. I sometimes wonder whether this was not his original complaint, and whether his whole story may not be rather apocryphal. (Mr. Whitelocke.)

No doubt this is an exceptional case; most of them have a considerable amount of discharge, and require at least a daily dressing. In fact they are in the condition of the previously recorded case of hydatid of the liver, or (only without the essential complication) of the next class to be considered—viz., tuberculous empyema. They may go on for many years, but are always liable to septic absorption, amyloid changes, osteo-arthritis, cerebral abscess, and such complications of prolonged supuration.

I now pass to the subject of *tuberculous empyema*.

Tubercle is the commonest cause of the non-closure of empyemata of moderate size. Sometimes this is because a calcareous gland, or a calcareous plate, or a piece of necrosed bone is hidden away in the depths of the cavity; sometimes apparently it is simply because the tubercle bacillus is present. Perhaps others can say something about the influence of tuberculin injections on the prognosis. I regret that I have as yet no valuable information to offer. Some tuberculous empyemata or empyemata in tuberculous subjects close after incision. I have already given one case. Here are some others:—

Dr. C., aged 40, had left pneumonia, followed in three weeks by empyema posteriorly, which was opened in February, 1896. There were physical signs at the left apex in front of doubtful significance. Tubercle bacilli were present in the pus of the empyema, and also for some time in the sputum. But he has made a perfect recovery, has left the profession, and lives an open-air life, to his own entire satisfaction. (Dr. Fairweather.)

Miss C., aged about 20, and said to be "intensely scrofulous," had pleurisy and pneumonia in 1882. She had no doubt had an empyema, which burst into the lung at that time, and externally, under the left breast, four years later, in

1886. I opened the cavity freely in 1887, which was followed by stoppage of the expectoration and great improvement of health. A posterior opening was made in 1888, and the whole thing closed in 1889, since which she has enjoyed very good health. It is now twenty-seven years since her first pleurisy. I have no positive proof that she was tuberculous; the family history suggests it strongly, but she goes back to the days before we were on such intimate terms with the tubercle bacillus as we are now. (Sir R. Douglas Powell.)

But even when the cavity remains open the patient may live for many years.

One patient, P., who was very tuberculous and whose right empyema formed copious calcareous plates, lived certainly more than twelve years after it was opened, and died at the age of 46 of acute pneumonia of the opposite lung. He lived a useful and happy life, in spite of his daily dressings, and the involvement of one kidney and testicle with tuberculous disease. (Dr. James Anderson.)

Mr. S., aged 42, had had phthisis for two years or more, when I opened a large right empyema in March, 1907. He has now a very large cavity, but a small amount of discharge. He has a good appetite and is well nourished, and leads an active, country life without at present showing any sign of amyloid degenerations. (Dr. J. K. Fowler.)

But, though these results are not uncommon, it is not safe to prognosticate too favourably for such people. It is fortunate, however, that one can honestly tell them, or at all events their friends, that a comparatively comfortable and possibly a prolonged life may be in store for them, and unnecessary to dwell upon the fact that an extension of the tuberculous process to other parts not infrequently occurs.

Estlander's operation is, in my experience, risky and seldom successful in tuberculous cases even if the cavity be small. Still, as it occasionally succeeds, one is sometimes tempted to offer the chance; but it is certainly not right to speak confidently about the result.

Perhaps the most remarkable case of empyema depending on the presence of tubercle which I have seen is that of Mr. M., aged 24, which may be thus summarized:—He had right brachial neuralgia in August, 1887, followed by left serous effusion, which resulted in an empyema, which healed slowly after two operations. About two and a half years after his first illness he had hæmoptysis, from which he ultimately died, and it was found that a calcareous gland had ulcerated both into the aorta and the trachea. (Dr. S. Phillips.)

Caries of the spine is not a common cause of empyema, and when the combination does occur the prognosis is certainly worse than that of uncomplicated Pott's disease. But even these cases are sometimes remarkably chronic.

One patient of mine, Major L., now aged 49, had pleurisy in 1889 (twenty years ago), and has survived numerous operations, including the evacuation of two huge gluteal abscesses that had tracked down from the spine. His empyema is still open, but he enjoys remarkably good health notwithstanding this inconvenience.

So much time has been devoted to disease of the pleura that I must pass on to two pulmonary conditions, and then bring my discourse to a close. And, first, with regard to *bronchiectasis*. I say nothing about bilateral cases, because they seldom, if ever, admit of surgical treatment. We have to deal with unilateral bronchiectasis, depending sometimes on the temporary or permanent lodgment of foreign bodies and sometimes on other preceding diseases of the lung or pleura. I will begin by reciting briefly a few cases showing how long they may go on and to what an extent improvement may take place:—

Master A. was aged 3 in 1887, when he inspired the vertebra of a rabbit whilst being fed with mulligatawny soup. I incised his lung in 1888, and he coughed up the bone in 1889. In December, 1895, he still had signs of a cavity at the right base, 1 oz. of mucopurulent expectoration daily, and clubbed fingers. The last account of him is that he is a good-looking young man and was married a year ago; that is twenty-two years after the catastrophe. (Dr. F. Hawkins and others.)

H. C. was admitted to University College Hospital in 1897, at the age of 17, with extensive bronchiectasis caused by the inspiration of the peg of a peg-top into the left lung. A year after the peg escaped through my incision. In 1901 he was still wearing a tube, and there was a good deal of offensive discharge and expectoration; he had had four or five attacks of hæmoptysis during the previous six months, and there was much clubbing of fingers. He is now said to be wonderfully well, is married and has two children, and is employed as a clerk on the railway. Further inquiries, however, show that the wound is still open and discharges about $1\frac{1}{2}$ oz. daily. He empties his cavity once in twenty-four hours, the amount being $1\frac{1}{2}$ oz. For the rest of the day he is free from cough. He wears the tube by day, but leaves it out at night. There has been no hæmoptysis for twelve months. The clubbing persists. There are still physical signs of a cavity. It is now twelve years since the accident. (Dr. Davies, of Port Talbot.)

Miss S., aged between 50 and 60, choked while eating Irish stew in October, 1904, and developed bronchiectasis at the base of the right lung with hardly any physical signs, but recurrent febrile attacks. These have ceased, but the cough and expectoration continue. She usually has more than one exhausting attack a day, followed by complete relief; if not, there is a constant nagging cough. She has occasional attacks of bronchitis. (Dr. S. R. Schofield.)

E. S. W. in February, 1891, when aged $6\frac{1}{2}$, inspired an ivory stud into the left lung, which was expectorated in the end of March. He developed bronchi-

ectasis, and when I saw him three years afterwards, in 1894, the physical signs were marked on the left side, but crackles were also to be heard on the right side. Since then I have not seen him, but I hear that he has grown up and, though he is rather pale and slight and not well developed, he is actively engaged in business which is partly manual, and that he flies about the country on a motor-bicycle. He has had very little medical attendance during the last ten years. There are still signs of a cavity, and he has to empty himself twice a day, but in the intervals has very little cough. Sometimes he gets a catarrh which pulls him down, and the last two or three springs he has taken trips to the Canaries or Egypt. There is still marked clubbing. He very frequently spits up some blood, but, as he is very reticent and never sees his doctor, it is impossible to say what this comes to. When asked how he feels, he always says "all right." It is eighteen years since the stud was inspired. (Dr. Anderson.)

F. J., at the age of 40, in 1890, coughed up a small quantity of bright blood without any effort while eating a damson as he was sitting by the fire after tea. He thought himself quite well at the time. He has, however, had a cough and expectoration ever since and attacks of hæmorrhage, at first slight and infrequent, but now more frequent and often most alarming. In 1902 the physical signs at the left base appeared most clearly to indicate the presence of a cavity, and, with Sir R. Douglas Powell's approval, I removed parts of several ribs over the spot, before exploring the lung, because the risk of hæmorrhage appeared so great. No cavity was, however, found, and at a subsequent puncture, when the patient was not anæsthetized, he almost died of hæmorrhage. He is still in the same condition; that is, he is well nourished and has a good appetite; but he has much cough and expectoration and frequent attacks of hæmoptysis, which are often alarming. He has to keep the house in the winter, but gets out in the fine weather, and has recently much benefited by a trip to Bournemouth. (Sir R. Douglas Powell and Mr. Tuxford.)

But I must not weary you with details of other cases, such as that of a lady, Mrs. B., now aged 44, whose trouble immediately followed a dental operation and who had hæmoptysis, often severe. At the moment she appears to have been cured by the removal of portions of several ribs over the affected portion of lung and the drainage of an abscess, and it is now eleven years since her illness began. (Dr. Lendon.)

Or of a lawyer, Mr. K. B., now aged 50, who after various operations on his nose developed bronchiectasis on the left side, and who also had serious hæmoptysis, and whose principal abscess I drained in 1903. There is now only a short, dry track lined by skin and mucous membrane by which his bronchus communicates with the external air. Over this he keeps a piece of plaster, and is only inconvenienced in arguing a case by the weakness of his voice. (Dr. P. Kidd.)

Or of that reported by Dr. C. T. Williams and myself in the *Transactions of the Royal Medical and Chirurgical Society* for 1886,¹ in which a cure was

¹ lxi, p. 317.

effected by operation; or of a recent similar case in hospital of a small boy who appears to be completely cured. (Dr. C. T. Williams.)

Or of a man, Mr. S., last seen in 1907, who was then aged 28, and whose trouble began when aged 2. He was then in fair health and talked of managing a sugar factory. (Dr. Trevithick.)

Or of a young lady, Miss B., now aged 25, whose trouble started twelve years ago with pleurisy, and who now enjoys moderate health, though there are still signs of a cavity at the right base and crackles are heard on the other side. (Dr. Brock.)

Trying to summarize my experience, it appears:—

(1) That patients with considerable bronchiectasis, whether operated on or not, may live for a great many years in spite of the continuance of the expectoration.

(2) That severe and recurrent hæmorrhage is a common symptom. But I have only known one patient die as a result of it, and that was an old man with a very septic state of things, whose lung I had freely incised.

(3) That in some cases, such as very localized ones or those caused by the inspiration of foreign bodies, drainage of the abscess may effect a cure, but more often attempts at surgery only lead to temporary relief. I have no personal experience of the removal of the affected portions of lung, but such operations are reported from Germany, and with a measure of success, and, it must be added, with an appreciable mortality.

(4) The risks of operation are septic absorption, including cerebral abscess and hæmorrhage.

It would be interesting to hear what is the chance of life in the purely medical cases. Of these I have seen a considerable number, but usually only once, and have had no means of following up their after-history. The late Dr. Schorstein¹ gives the cause of death in sixty-three patients who were admitted to the Brompton Hospital between 1882 and 1904. This does not help much in the matter of prognosis, because he does not give the duration of the disease, and, of course, we have no account of those people who endure bronchiectasis—say, like the gout—as one of the accidents of life, and who do not come into hospital to die of it, and, in fact, perhaps die of old age or some other complaint. There are, however, three points of much interest: one is prepared to find that the largest number of deaths—seventeen—were from bronchopneumonia, but it is startling to see that thirteen died of cerebral abscess, and instructive that four died of hæmoptysis.

In speaking of *localized pulmonary abscess*, we are again not concerned with those cases which die outright, whether opened or not, such

¹ *Lancet*, 1909, ii, p. 843.

as some of those which occur during an acute pneumonia or some secondary septic cases. Nor need we consider the ordinary tuberculous apical vomicae. The point of interest now is the after-history of such localized abscesses as arise in connexion with pneumonia, or embolism, or the bursting into the lung of abscesses from other parts, or the presence of foreign bodies and a very small number of tuberculous cases. I think I have only seen one basic tuberculous cavity close after incision. The others have benefited by operation only in so far as the expectoration was diminished. Otherwise the disease has followed its natural course. I have seen several cases where what was diagnosed as small pulmonary abscesses, which exploration failed to discover, were expectorated, and this was followed by apparently complete recovery. And I can produce the histories of several cases where, when a localized pulmonary abscess has been successfully reached, cure has followed, and the patient has remained well for many years. I believe, indeed, that such patients are no worse off than they were before. Here are some examples:—

Mrs. C., aged 43. An operation was performed on the nose in November, 1899. Very soon afterwards cough set in and there was a disagreeable taste in the mouth. The following month a foetid expectoration began with symptoms and signs of gangrene of the anterior and upper part of the left lung. I opened an abscess near the apex of the lung in June, 1900, and in October had to reintroduce the tube. The abscess closed in the course of 1901. She has enjoyed the best of health since—that is, ten years. (Dr. Booth.)

Mr. D., aged 26, had an abscess of the liver unsuccessfully explored in May, 1899. Expectoration of pus like liver pus followed in July. Before I saw him he had had both pleurae and both subdiaphragmatic spaces explored. I found an abscess in the left lung at the angle of the scapula and opened it in March, 1900; the wound was healed by November 27, 1900. He is now perfectly well, having spent four and a half of the intervening nine years in India, and being now in the active pursuit of his profession. (Dr. John Anderson.)

Dr. S., aged 46, had enjoyed excellent health in India with the exception of an occasional attack of ague. He had "influenza" in March, 1896, was much exposed in the jungle on a shooting expedition in April, had a rigor on May 1, and from that date till he was seen by Dr. Bruce and myself in September he always had a high evening temperature. On May 5 he expectorated some pus, and this continued and became foetid, containing black pieces in which were found very long rod-shaped bacilli. He had an abscess in the apex of the right lung, which I opened on October 9. The temperature came down gradually, but he was healed in the early part of 1897, and sends me annually at Christmas-time a report of his excellent health, in which his only complaint is that he gets too stout in spite of much exercise in a hot climate. (Dr. J. Mitchell Bruce.)

Mrs. D., of whom I have no notes, had for years occasional attacks of fever accompanied by very offensive expectoration, small in amount, which appeared to come from the base of the right lung. I explored very thoroughly, but could not find it. It is now several years since there has been any attack of the sort, and she has apparently quite recovered. It should be added that she had one or two attacks much resembling appendicitis, and it is possible that this may have been the source of the pus. (Dr. Voisey.)

The cautious prognostician will not forget that it is not only in the case of serous effusions but also in that of empyemas and pulmonary abscess that *carcinoma* and *actinomycosis* have to be thought of.

I have opened a pulmonary abscess in a man aged 64 (Mr. L.) and failed to discover till many months afterwards that it started from a carcinoma (Dr. Brook); and I have treated an empyema in the Brompton Hospital till the patient died with cerebral symptoms, which were naturally supposed to be due to abscess, but really depended upon secondary malignant growths in the brain, the primary tumour being in the thorax.

As to *actinomycosis*, it is a fruitful cause of error, and nothing but great watchfulness will prevent mistakes, which not only endanger the chances of the patient, but the reputation of the doctor.

Perhaps I shall be thought to be giving too hopeful an account of the probable future of patients after a pulmonary abscess. It is impossible to say that some cases of bronchiectasis, following obscure pulmonary conditions, may not depend upon such a cause; but I have no facts to offer pointing in this direction.

I wish I could give any guide as to the likelihood of the occurrence of *cerebral abscess*, which so comparatively often steps in to blast the most hopeful prospects. I have seen it occur in what appeared to be ordinary straightforward cases of empyema, in bronchiectasis, both acute and chronic (but only after operation), in gangrene of the lung, and in several cases of abscesses of the lung caused by the bursting upwards of an abscess of the liver. In the *Lancet* of September 18, 1909,¹ is published a valuable contribution to this subject by the late Dr. Schorstein, in which he gives details of nineteen cases of cerebral abscess, fourteen of which were associated with bronchiectasis, and of these only five had been submitted to operation. With all its interest the paper does not help us in forming a prognosis, except in so far as it suggests that, of all the diseases under discussion, bronchiectasis is the most likely to be followed by cerebral abscess. We must not, however, accept the proportion fourteen out of nineteen as not open to doubt, for it must be remembered that secondary pulmonary abscesses, such as those

¹ ii, p. 843.

starting in abscess of the liver, do not often find their way into the Brompton Hospital. As is well known, those abscesses are often multiple and often difficult to localize. I have, however, opened several, but so far without a single success. One does not mention this bugbear in giving a prognosis, but it is a shadow in the background for those who have much to do with thoracic surgery, making them anxiously examine the pulse and temperature of any such patient who unexpectedly complains of a headache. Cerebral abscess is the end of all prognosis, and it may supervene after many years. I do not say that this last case I will quote is an example, as the complicated problem was neither cleared up by an operation nor an autopsy:—

Mrs. S., aged 19, had left pleurisy during typhoid fever, which was followed by an abscess that burst in the axilla, and which certainly came from inside the chest, though Sir James Paget thought it might have arisen from typhoid necrosis of a rib. I operated on it seven and again nine years later, in 1884 and 1886, but it never healed. In 1890 she struck the right side of her head against the iron bar by which a shop-lamp was suspended, and she was insensible for a quarter of an hour. In 1897 ovariectomy was performed. In 1898 she had a sudden seizure, followed by cerebral symptoms. There was much pain at that part of the head which had been struck eight years before, and the left arm became paralyzed; but she refused operation and died three weeks afterwards, but without signs of coma, of cerebral abscess, as it was thought. She was then aged 38, and had survived her attack of pleurisy nineteen years. (Dr. Smith.)

"What an unscientifically and unsatisfactorily reported case!" it may be said. "What a striking illustration," I reply, "of the difficulties that beset the conscientious prophet!" How many life-histories, even when properly studied to the end, are so complicated that they do not help us in our auguries! So much, indeed, is this the case that one is sometimes tempted to give up the calling in despair; and yet it has its fascinating side, especially for those of us who are no longer in the heyday of youth; because, while those who are, maybe, better fitted for striking out into new fields, it is absolutely impossible for them to have gathered the facts which will entitle them from their own knowledge to admission into the "goodly fellowship." I would therefore encourage my contemporaries and senior colleagues to add as only they can do to the interest and value of our meetings by bringing us their matured conclusions, and to impress upon my juniors the importance of careful note-taking, including records of the abodes of their patients and the doctors who attend them, so that when they (as in a few short years they will do) step into our shoes their "old experience" may "attain to something like prophetic strain."

Surgical Section.

December 14, 1909.

Mr. G. H. MAKINS, C.B., Vice-President of the Section, in the Chair.

The Diagnosis and Treatment of Duodenal Ulcer.

An Address Introductory to a Discussion on the Subject.

By B. G. A. MOYNIHAN, M.S.

THERE are few diseases whose symptoms appear in such a definite and well-ordered sequence as is observed in duodenal ulcer. It is true that there are cases, of which fuller details must presently be given, in which the regular appearance of the symptoms is absent, or in which one symptom is so exaggerated as to dwarf, or even destroy, the value of others. But these exceptions are few, and they do not belittle the value of the general statement that the symptoms of duodenal ulcer are definite and not easily to be mistaken, and that they appear in an order and with a precision which are indeed remarkable.

The patient may date his complaint from an early period in his life. It is not very uncommon for a man, in answer to the question as to how long he has suffered, to reply "All my life." A man of 61, upon whom I operated, had first experienced symptoms at the age of 19; others had symptoms "as long as they could remember." This goes to show that the ulceration may begin at an early period in life, and the symptoms may, with periods of repose, continue up to middle age, or even to advanced years. As a rule the patient is in middle age, from 25 to 45; and males are more frequently affected than females. If the earlier history is well remembered, the patient will say that insidiously, almost imperceptibly, he began to suffer from a sense of weight, oppression, or distension in the epigastrium after meals. At the first the discomfort may apparently be capricious, but it is not long

before notice is taken of the fact that it comes usually two hours, or a little more, after food has been taken. Immediately after a meal there is ease: if pain and discomfort were present before, the meal relieves them, and banishes them completely for a time. Then, again, the pain is felt in two hours, three hours, four hours, or sometimes even six hours later. As a rule the pain comes gradually, and gradually increases, becoming more severe and being accompanied by a sense of fullness, distension, a "blown-out" feeling; there is an eructation of bitter fluid or of gas, which affords relief. The interval between the taking of food and the onset of the pain is very remarkable; it is constant from day to day if the character and quantity of the food remain the same. If the food is entirely liquid, the pain comes rather earlier; if it is heavy, solid, "indigestible," the pain comes later; with an ordinary meal of liquid and solid, the pain very rarely appears in less than two hours. Many patients will volunteer the statement that the pain begins to appear "when they are beginning to feel hungry," and I therefore suggested in one of my early papers the term "hunger pain" as descriptive of this particular symptom. The pain, as a rule, is noticed at first only, or chiefly, after the heaviest meal of the day. If dinner is taken between 1 p.m. and 2 p.m., the pain will come with unvarying regularity at, or near, 4 p.m. For a long period this may be the only time of day when discomfort is felt, but later in the attack, or in subsequent attacks, it is noticed that after every meal the pain comes at its characteristic interval, that by every meal the pain is relieved, only to return in due time. When inquiry is made from a patient as to whether food causes the pain, he will not seldom answer, "Oh, no! Food always makes the pain better; the pain comes when I am beginning to feel hungry."

It is a very characteristic feature of the pain that it wakes the patient in the night, and constantly the time of the waking is said to be 2 o'clock. The relief of the pain by food, quickly realized by patients themselves, leads to the practice of keeping near at hand a biscuit, or some other food or drink, which can be taken at once. Many patients carry a biscuit in the pocket, or have a glass of milk and a piece of bread and butter ready at certain times, to be taken at the moment of the onset of pain. It is a common experience to find that patients place by their bedsides some food to be taken in the early hours of the morning when the pain awakes them. The regular appearance of the pain after definite intervals from the taking of food is remarkable and is consistent. The pain is often preceded

or accompanied by a sensation of weight or of fullness and distension in the epigastrium; it is described as "boring," "gnawing," "burning." It may be relieved by belching, and constant efforts are often made to bring about the eructation of gas, which is followed by momentary relief. Sometimes there may be a slight regurgitation of food, and the patient complains that the taste of this is bitter, or acid; the throat then feels hot or as if scalded, and the teeth are said to feel as if made of chalk. A few patients complain that when the pain is beginning to develop, a free gush of saliva may occur. The swallowing of this may give temporary relief to the pain. In some cases the flow of saliva may be copious and distressing.

For long periods, sometimes throughout the history of the case, the pain remains confined to the epigastrium, but it may strike through to the back or pass round the right side. When the pain is severe, relief is often gained by pressure, and I have known patients, wakened in the night, to hug a pillow to the abdomen to obtain relief in this way. On some occasions, though this is infrequent, the pain is said to be "cramp-like" in character, a sort of spasm is felt with exacerbations and remissions, as in all forms of "colic." It is very probable that a spasm of the pylorus, protective no doubt in its character, is actually present; for such a condition may, as I pointed out several years ago, be witnessed during the conduct of an operation. The pyloric muscle hardens by degrees until a state of firm contraction is reached, when a solid cylindrical whitish mass forms, which imparts a feeling similar to that experienced when the normal uterus is handled. The spasm slowly passes away, and the stomach assumes its normal appearance. This sensation of cramp is often accompanied by a feeling of great epigastric distension. I have twice seen this cramp well marked in patients who were operated upon under local anæsthesia only; they both described their sensations at the moment as one of "bursting and distension."¹

Throughout the whole period during which the pain is felt the appetite remains good. In many cases the patient volunteers the statement that he feels a keen relish for his food, takes it with good appetite, and enjoys it. Frequently he eats less than he feels he would enjoy because experience has taught him that excess, or even satisfaction, is apt to be followed by an increase of pain, or pain of a more

¹ I have often wondered, since this, whether the "colic" described by patients (hepatic colic, renal colic, intestinal colic) is due, as we have always supposed, to the contraction of unstriped muscular tissue; it is possible that it is rather the result of the distension which occurs behind the segment in which the muscular spasm is occurring.

enduring character. Fluid food, when taken to the exclusion of all solids, often causes the pain to come earlier after food and to last longer than when the ordinary meals are taken. A patient will often say that he feels worse when he is strictly dieted in this way, but, as a rule, persistence in liquid diet, especially during the earlier stages of the disease, will bring relief after a time. Vomiting is very infrequent; it is rarely present until stenosis develops, and stenosis appears only in the later periods, when the ulcer or ulcers are healed. The majority of the patients upon whom I have operated have never vomited.

These are the characteristic symptoms described by the patient in the anamnesis. Upon them alone a confident diagnosis of duodenal ulcer may be made. Perhaps the most characteristic feature of duodenal ulcer is the periodicity of the symptoms and their recurrence from time to time in "attacks," their complete abeyance in the intervals. A patient who has suffered for years will say that an "attack" comes on as a result of exposure to cold, or getting the feet wet, or a hasty or "indigestible" meal, or worry or overwork. A cause can almost always be assigned for the onset of symptoms; a recurrence of the cause is always followed by a reappearance of the symptoms. The most common of all these causes is "getting cold"; in consequence, the great majority of the patients will say that the attacks are especially prone to come in the winter months—December, January, or February. In the summer the symptoms are almost always absent. One patient of mine was perfectly well for three years when in India. He returned to England in November, and within a fortnight had "caught a chill" and all the symptoms returned. The "attacks" vary in length from two to three weeks up to several months. It is remarkable that an attack may frequently be cut short by a few days' rest in the country or at the seaside. Two of my patients, medical men, told me that a "long weekend" at the sea, with plenty of open-air exercise, free from the anxieties of practice, would always cut short an attack in the earlier years of their trouble. In the end the pain may become a matter of daily experience, but it still retains its characters as I have described them. In the intervals between the attacks there is complete immunity from suffering: food is taken with full enjoyment and with keen zest; there is no discomfort of any kind; weight is gained, and mental and bodily vigour are at their highest. So complete may the recovery be that the very suggestion that the former attacks have been due to organic disease may be scouted or received with the tolerant smile of disbelief. The explanation is given that there has been "hyperchlorhydria," or that

the case is one of "acid dyspepsia," or possibly of "neurosis." The idea is deep-rooted that the symptoms are due to an excessive acidity in the gastric juice, but, as I shall presently show, there is no foundation in fact for this venerable fallacy.

These symptoms, so perfectly characteristic of duodenal ulcer, may be present for years without producing any physical signs. It is therefore not necessary to the attaining of an accurate diagnosis that any examination of the patient be made. The anamnesis is everything, the physical examination is nothing. There is, in the stage when the presence of the ulcer should be recognized, no single physical sign indicating the presence of organic disease. Signs which confirm the accuracy of the diagnosis may appear later, but there is no need to await their arrival before making, as we can with the utmost confidence, an exact diagnosis.

In a rather later stage dilatation of the stomach, with motor incompetence, may appear; the stomach, that is to say, is unable to empty itself completely, within the normal period of time. What would be considered a "normal period" is not agreed upon by all writers. I have arbitrarily adopted the period of twelve hours. If a stomach is not able to empty its contents into the duodenum within twelve hours, it is very probable that there is organic disease which prevents it doing so. Gastric stasis, then, may be found in cases of duodenal ulcer; it is due always to the narrowing which occurs by reason of the healing—partial as a rule, but sometimes complete—of the ulcer or ulcers. I have never found that stasis of this degree was present as a result of pyloric spasm. It is possible that a spasm of the pylorus prevents the stomach from emptying as quickly as it otherwise would do; for the spasm no doubt exists because of the need for protection of the ulcer of the duodenum from the harm which contact with the acid chyme would inflict. The spasm is reflex and is protective, as was so beautifully shown by Cannon and Murphy.¹ But a spasmodic contraction of the muscle which guards the outlet does not prevent the stomach from emptying within the period of twelve hours. Its occurrence is probably protective also in the fact that it arouses symptoms the mere presence of which makes the patient less eager to take food in full quantities. Gastric stasis denotes therefore the existence of a narrowing in the duodenum due to organic disease. When this narrowing attains even a very moderate degree, an hypertrophy of the musculature of the stomach develops, as always happens in the alimentary canal; and the evidence of this may

¹ *Annals of Surgery*, Philad., 1906, xliii, p. 512.

be found in the peristaltic waves visible when the stomach is examined. If the stomach is empty or only partially filled these muscular contractions may not be seen, but the administration of the two halves of a Seidlitz powder separately will soon excite them to appear.

In all cases of duodenal ulcer, indeed in all cases of intractable stomach disorder, a test-meal should be given. There are a number, by no means inconsiderable, of patients who have been referred to me as cases of "hyperacidity," "acid gastritis," upon whom I have operated and have demonstrated the existence of a duodenal ulcer. Recurrent severe "hyperchlorhydria" is duodenal ulcer. The symptoms of which the patient makes complaint are ascribed to hyperacidity; but it is extremely interesting to know that it is usual for the gastric juice in such cases to be normal: there is no hyperacidity at all.

It is the rule in intractable cases of so-called "acid dyspepsia" for there to be no hyperacidity; and it is in my experience invariable to find duodenal ulcer in such cases. No one is more sceptical than myself in interpreting the appearance of the stomach and duodenum during the course of an operation. I do not believe in the duodenal ulcer which I cannot demonstrate to the most sceptical assistant or onlooker. No "anæmic spots" or trivial, often imaginary, thickenings do I accept as being enough to account for protracted symptoms. The ulcer is always a visible, tangible, demonstrable lesion; and in a large experience I have never operated upon a case of protracted and recurrent "hyperchlorhydria" without finding a duodenal ulcer. In such cases it is rare to find any excess of acidity when a test-meal has been given. A sign which sometimes appears early in the course of this disease, but which is more often a late symptom, is hæmorrhage. It is, of course, an evidence that the process of ulceration has extended to such a depth as to open up a large vessel, and so deep an invasion of the coats of the bowel is usually accomplished only after the lapse of months or of years. When bleeding occurs in quantity sufficient for it to be recognized as hæmatemesis or melæna, it is an evidence of the deep penetration of the walls of the duodenum by an ulcer whose existence should have been recognized long ago. Neither hæmatemesis nor melæna should be considered as among the usual signs of duodenal ulcer; they are both complications whose onset should have been forestalled; they are a witness to neglected opportunities.

The frequency with which bleeding occurs from a duodenal ulcer has been variously estimated by different authorities. Thus Kraus in the 70 cases collected by him found that in 20 free hæmorrhage had

been observed. Oppenheimer in "over 100" cases found bleeding recorded in 34. In Perry and Shaw's series of 60 cases presenting symptoms in a total of 151 cases, hæmatemesis or melæna was present in 23. Nine patients had hæmatemesis, 9 had melæna, and 5 had both hæmatemesis and melæna. In Nothnagel's "Encyclopædia" (p. 245) it is said that "severe hæmorrhage occurs in about one-third of cases." Fenwick estimates the frequency of hæmorrhage in acute cases at 26 per cent., or in chronic cases at 40 per cent.

All these figures seem to me to be valueless. They are compiled from statistics every item in which is open to disproof or doubt. The symptoms which characterize duodenal ulcer so unmistakably were unknown to every one of these authorities; the frequency of the disease was therefore quite unappreciated. Only patients who suffered from such complications as stenosis, perforation, or hæmorrhage were known to suffer from an ulcer in the duodenum, and the verification of the diagnosis could only then be made upon the post-mortem table. In my own series of cases hæmorrhage has been noticed in 70. But, with the new light which now has been shed upon this important subject by the work of the surgeon, we have come to recognize that hæmorrhage is not a symptom but a late complication, that its onset is not to be awaited in order that a doubtful diagnosis may receive confirmation, but that its appearance is to be prevented by a timely recognition of the significance of the early symptoms. Hæmorrhage, when it does occur, may be manifest either as hæmatemesis or as melæna; the blood may be discharged in the vomit or in the fæces. Melæna may, and indeed usually does, exist without hæmatemesis; but when blood is vomited there is almost without exception some blood also in the stools. I believe hæmorrhage from a duodenal ulcer to be a sign of grave significance, of far more serious import than bleeding from a gastric ulcer. In the latter death very rarely occurs; in the former it is more frequent than is generally supposed. I have thrice had the experience of advising operation for duodenal ulcer in cases where hæmorrhage subsequently occurred and proved fatal before surgical help could be given. Hæmorrhage from a gastric ulcer is sometimes very copious and gives rise to great alarm, but when the bleeding ceases spontaneously the patient recovers quickly. In duodenal ulcer the bleeding causes faintness and anæmia, the exact origin of which may not be obvious till the bowels are moved. Then faintness and prostration come again and again, an abundance of blood—at first black, but later of a brighter hue—is passed, and the patient may rapidly become exsanguine and die.

The manner in which hæmorrhage appears varies much in different cases. As a rule, there is a considerable exacerbation in the symptoms before the bleeding comes; the "indigestion" is more acute, the feeling of distension or oppression after food is greater, and the patient himself does not feel so well. Then suddenly he feels faint, and weak and breathless, the head feels "light and swimming," and the sight seems quickly to grow dim. The patient looks white, his lips are bloodless, and sweat covers the brow; he asks constantly for air and is breathless; he displays, in brief, all the classical signs of an internal hæmorrhage. That this has occurred is presently made certain by the voiding of blood in the characteristic "tarry" motion, or by the ejection of brighter blood in the vomit. In other cases the hæmorrhage may occur insidiously, without the patient having noticed it; he is aware only of a continuing weakness and frailty which he can hardly understand. A case in my own series was an exemplary instance of this. The man was sent to me because of a right inguinal hernia. As he entered my room I was struck with his blanched appearance, and my diagnosis of duodenal ulcer was made before he reached a chair. When I asked him to tell me his symptoms, he had nothing to say but that he had a hernia. I asked if he had noticed any loss of blood; he replied in the negative. I inquired whether "indigestion" had been observed, and he said at once that he had "suffered from that for years" and that recently it had been very severe. I took the man into hospital, and found that he had melæna. I elicited then a perfectly clear history of duodenal ulcer. At the operation I found a large chronic ulcer, for which I performed gastro-enterostomy. The case has been very successful.

It is probable that a certain degree of hæmorrhage occurs in many cases of duodenal ulcer without being recognized. The surface of the ulcer, when fretted, probably bleeds a little, and if the stools were carefully and regularly examined traces of occult blood would surely be found. I have in a few cases found this to be the case, but since I have realized how accurately the existence of an ulcer can be recognized from a study of the clinical symptoms alone, I have not pursued this line of investigation closely. Occult blood—blood, that is, in quantities too small to be seen by the naked eye but capable of recognition by other tests—is therefore probably not very infrequent. The vessels which are opened by the deep invasion of the walls of the bowel by the ulcer vary considerably in size; in proportion to their size the hæmorrhage is slight or abundant. The following are some of the larger vessels which have

been eroded, with the result that fatal hæmorrhage has occurred: the aorta, the hepatic, gastro-duodenal, superior pancreatico-duodenal, right gastro-epiploic, and pyloric arteries; the portal and superior mesenteric veins. In a few cases hæmorrhage has been so sudden in onset and so profuse as to cause death, which was almost sudden. When the base of an ulcer from which fatal hæmorrhage has occurred comes to be examined, the vessel involved is usually found to have thick and rigid walls. The opening from which the blood has come is at the side of the artery, which remains widely open. There is neither closure nor retraction of the vessel, whose walls, stiff as the stem of a clay pipe, seem incapable of contraction.

Such is a brief description of the characteristic symptoms of chronic duodenal ulcer. If a patient presents these symptoms, the diagnosis of duodenal ulcer may confidently be entertained. There is no need for further evidence than that which is so afforded. I constantly operate upon the strength of the history alone, and as often do I demonstrate the existence of a chronic ulcer, a tangible and visible lesion, as the cause of the symptoms. Of nothing concerned with the relationship between altered structure and altered function am I so convinced as that symptoms such as I have portrayed owe their origin to, and are dependent for their perpetuation and their periodic repetition upon, a chronic duodenal ulcer.

TREATMENT.

In my opinion the treatment of a chronic duodenal ulcer should always be surgical. Chronicity in an ulcer is attested by the recurrence of attacks of a well-defined character. When a series of these attacks have occurred the ulcer is always to be plainly seen and demonstrated; and my experience in a long series of operations is that the conditions in the ulcer, as a rule, are such that nothing but surgical treatment could possibly avail. The ulcer is so large, or so indurated, or the ulcers are so numerous that, even if the lesions were to cicatrize completely, one of two things would result: either a hard, fibrous surface, readily breaking down under provocation, would remain, or a stenosis of the bowel would inevitably follow.

But the question at once arises as to when the case is first to be recognized as surgical; as to when we are entitled to say that medical treatment will probably prove to be of little or no permanent value. What are the conditions present in the duodenum in the first of all the

"attacks"? I have at present no adequate means of knowing. I have only once operated in or after a first attack, and so have little evidence upon the point. In this case the pathologist reported that a small fibrous scar was present in the small piece of the duodenum which I had excised. I was myself very doubtful whether an ulcer had been present. I have once operated at the close of a second attack; the patient being referred to me by my colleague, Dr. T. Wardrop Griffith. An ulcer about $\frac{1}{4}$ in. in diameter on the anterior surface of the duodenum was excised. The ulcer was clean, with terraced margins, indurated, and it had destroyed the whole of the muscular coat of the bowel. Though the attack had passed away completely, the ulcer was still open and unhealed. In subsequent attacks the ulcer is always to be seen, and possesses the characteristic appearance. An ulcer which has caused recurrent attacks has always involved the serous coat, and is accordingly easily to be seen from without. It is only when attacks recur that a diagnosis of chronic duodenal ulcer can confidently be made; it is only when this diagnosis can be made that surgical treatment is necessary. In a first attack, or even in a second, medical treatment may be tried. But I believe it to be true to say that the significance of the symptoms in these attacks has never yet been fully recognized by the physician. It has not been realized that these symptoms are due to a structural lesion, and consequently (after a diagnosis of "acid gastritis" or "neurosis") treatment has been perfunctory and brief. Up to the present time it is, with the exception of the single case I have mentioned, only after repeated attacks, sustained often over a period of years, that the surgical needs of the case have been recognized. If the first of the attacks be due to a duodenal ulcer, then medical treatment of a sufficiently protracted and careful character should be tried. But when attacks recur in the typical manner I have described, the lesion found is of such a nature that anything other than surgical treatment is not worth considering. It is safer, speedier, and more certain than any other mode of treatment.

The surgical treatment of duodenal ulcer may be carried out by—

- (1) Excision of the ulcer and restoration of the duodenal canal.
- (2) Excision of a cylinder of the duodenum by closure of the distal end and union of the pyloric cut end with the side of the second portion of the duodenum.
- (3) Partial resection of the duodenum, followed by closure of both cut ends and gastro-enterostomy.
- (4) Gastro-enterostomy.

As the cases are seen now, the performance of gastro-enterostomy is almost always necessary. The conditions of the ulcer are such that a restoration of the duodenum to its normal state is impossible, and all that can safely be done is to afford a new outlet from the stomach. It is now my invariable practice to infold the ulcer, in precisely the same manner as if a perforation had occurred. This allows the ulcer to heal more speedily, as it ensues that almost no food passes through the pylorus.

ANALYSIS OF 197 CASES OF DUODENAL ULCER OPERATED UPON BY MR. MOYNIHAN (1900-1908).

Prepared by HAROLD COLLINSON, M.S.

PERFORATING DUODENAL ULCER.

THERE are eleven cases in which operation was performed for perforation of a duodenal ulcer.

Sex.—Of these cases seven are males and four females.

Age.—The youngest patients were aged 17 at the time of operation and both were females; the oldest was aged 44. The average age was 29 years.

Previous History.—In all the cases there was complaint of previous indigestion, varying in duration from four weeks to many years. In two cases there had been attacks of hæmatemesis, but in none had melæna been noted. In three cases the perforation appears to have been precipitated by injury or exertion. Case 2 was climbing a ladder when seized with acute pain. Case 4 was straining heavily at work. Case 8 received a blow on the left side of the abdomen two hours before admission. In Case 10 an early duodenal ulcer was apparently caused to perforate by extreme distension of the intestine behind an acute obstruction in a right duodenal hernia.

Period between Perforation and Operative Treatment.—The shortest interval was four hours; the longest in the acute perforations was three days, and in the subacute cases three weeks.

Subacute Perforations.—In three cases (5, 6, and 7) the perforation was subacute, and the cases were operated on four days, four days, and three weeks after perforation respectively.

Results.—There were eight recoveries and three deaths. The three fatal cases (1, 8, and 11) were operated on twenty-six hours, three days, and twenty-six-and-a-half hours respectively after perforation. Gastro-enterostomy was performed at the same time as the perforation was closed in four cases (1, 6, 9, and 10); of these cases the first died. In Case 3 a gastro-enterostomy was subsequently necessary on account of stenosis. In Case 4 a subphrenic abscess developed, and was opened and drained on the twentieth day.

CHRONIC ULCER.

The patients number 186, and the period covered is nine years (1900-8).

Sex.—Males: 137, or 73·6 per cent.; females, 49, or 26·3 per cent.

Variety of Ulcer.—Amongst the 137 male patients there were 107 cases in which duodenal ulcer alone was found; 30 cases in which both gastric and duodenal ulcers were present. Of the 49 female patients, 32 had duodenal ulcer alone, and 17 both gastric and duodenal ulcers. The proportion of males to females amongst the cases presenting duodenal ulcer only was therefore more than three to one. In the first 40 cases operated upon, evidence of both gastric and duodenal ulceration was found in 24.

Age.—The distribution of the patients in the various decennial periods is as follows:—

Years	1 to 10	0
"	11 " 20	3
"	21 " 30	37
"	31 " 40	56
"	41 " 50	45
"	51 " 60	27
"	61 " 70	11
Age not stated		7

The youngest was aged 17 and the oldest 67; it must be borne in mind, however, that the age given is that of the patient at the time of operation and not at the onset of symptoms, and that many of the patients who were over 40 had had symptoms for a considerable number of years. The longest duration of illness before operation was forty years (Case 163), and the shortest seven weeks (Case 45); in this case melæna was severe and practically the only symptom.

Symptoms.—In examining the case-histories one is impressed by two facts: firstly, that in the earlier cases the clinical picture which we are now accustomed to associate with the presence of duodenal ulcer is only imperfectly indicated in the account of the patient's symptoms, whilst in the majority of the later cases the patient's account of his symptoms is typical; the second point of interest is that, at first, operation was in a large proportion of the cases undertaken for the more serious results of duodenal ulceration rather than for the earlier symptoms. The details of the cases mark the gradual increase of our knowledge of duodenal ulcer; in the earlier period symptoms were little understood, accurate information as to the time of onset of pain was not sought, and it was only the grosser and more serious results of ulceration which brought the patient into the hands of the surgeon; as our knowledge and familiarity with the condition have increased, so the cases have been seen earlier, their symptoms more carefully investigated, and operation advised and performed in most cases before the onset of dangerous complications.

Hæmorrhage.—Seventy patients (37·6 per cent.) gave a history of bleeding at one time or another; of these, seventeen had hæmatemesis alone, twenty-three had melæna alone, and thirty had both hæmatemesis and melæna. Amongst the 139 cases in which duodenal ulcer alone was found, hæmorrhage

was noted in forty-nine cases (35·2 per cent.), nine had hæmatemesis alone (6·4 per cent.), eighteen had melæna alone (13 per cent.), and twenty-two had hæmatemesis and melæna (15·8 per cent.).

Perforation of Ulcer.—Amongst the 139 cases in which duodenal ulcer was found alone, there were evidences of perforation in five (3·6 per cent.) Case 17: Operated upon eight months previously for perforation. Cases 38 and 177: In these, a subacute perforation occurred a short time before. Cases 169 and 178: An ulcer had probably perforated a considerable time previous to the operation. In addition to the above cases, a perforation had probably occurred some time before in Case 98, but it is uncertain whether it was of a gastric or duodenal ulcer. Case 147 showed an old perforation of a gastric ulcer.

Tetany.—Three cases (6, 10, and 58) gave a history of tetany. In all of these both gastric and duodenal ulcers were present and the stomach much dilated.

Cardiospasm.—In three cases (161, 168, and 184) more or less spasm of the cardiac opening of the stomach was present, and in Case 161 this was, apparently, the chief trouble.

Operative Treatment.—Upon the 186 patients 193 operations were performed; seven patients were operated upon twice. The 193 operations may be classified as follows:—

Posterior gastro-enterostomy by simple suture	...	78
" " " " with infolding of ulcer	...	84
" " " " with Laplace's forceps (Cases 1 and 2)	...	2
" " " " with Murphy's button (Case 3)	...	1
" " " " by Mayo's method (Cases 74, 84, 106, 152)	...	4
" " " " and gastroplasty (Case 108)	...	1
Anterior " " with lateral anastomosis (Case 159)	...	1
Modified Roux's operation (Cases 17, 169)	...	2
Division of loop and lateral implantation (Case 100)	...	1
Lateral anastomosis between limbs of loop (Case 40)	...	1
Posterior gastro-enterostomy with cholecystotomy (Cases 12, 78, 83, 130)	...	4
" " " " with cholecystectomy (Cases 161, 163)	...	2
" " " " with appendicectomy (Cases 75, 176, 189)	...	3
" " " " with radical cure of hernia (Case 46)	...	1
" " " " with excision of duodenal ulcer (Cases 114, 182)	...	2
Excision of duodenal ulcer alone (Case 183)	...	1
Excision of gastric ulcer (Case 173)	...	1
Closure of pylorus (Case 172)	...	1
Cholecystenterostomy (Case 180)	...	1
Cholecystectomy (Case 80)	...	1
Exploratory laparotomy (Case 83)	...	1
Total	...	193

In Cases 1 and 2 gastro-enterostomy was performed by the aid of Laplace's forceps, but with strikingly different results: the former of the two patients is now well and free from trouble, the second returned two months later with a recurrence of symptoms. Then the abdomen was opened (Case 3) the stoma was found to be almost closed, and a fresh anastomosis was performed with Murphy's button; the patient is now quite well. These three operations are the only ones in which any appliance was used in the performance of the anastomosis. In four cases posterior gastro-enterostomy was performed

82 Moynihan: *Diagnosis and Treatment of Duodenal Ulcer*

by the antiperistaltic, Mayo's, method; the number is too small for one to draw any conclusions as to the relative advantages of this method, but one of the four patients has been troubled with regurgitant vomiting since the operation. The other cases, which were twice operated upon, are of considerable interest.

Case 24 (see also Case 40): A posterior gastro-enterostomy was performed by simple suture; one week later severe vomiting commenced, and lasted, with short intermissions, for a year, when the abdomen was reopened. It was then found that the whole of the small intestine, with the exception of the last 18 in., had herniated into the lesser peritoneal sac through the opening made in the transverse mesocolon. The herniated bowel was reduced, and a rather long loop was found to have been left between the flexure and the stoma; a lateral anastomosis was performed between the limbs of the loop and the margins of the opening in the mesocolon sutured to the line of the gastro-enterostomy opening. A similar accident produced a fatal result in Case 16.

Case 54: In this case there was no stenosis, and the duodenum was not infolded at the time of the gastro-enterostomy; for a time the operation conferred some benefit, but he returned three years later (see Case 172) with a recurrence of all symptoms, pain two to three hours after food (relieved by food or bicarbonate of soda), and frequent vomiting. The abdomen was reopened, and the following condition found: The anastomosis showed a slightly longer loop than would be left at the present time, and the stoma would admit three fingers easily. The pylorus was patent, and at the site of the ulcer found at the previous operation, of which a drawing had been kept in the old notes, a large scar was present; close to this were two other well-marked ulcers, evidently of recent date. The explanation seems to be that pyloro-spasm was formerly present along with the duodenal ulcer; the gastro-enterostomy acted at first, but as the spasm relaxed the opening ceased to be functional, and ulceration recurred. The pylorus was closed by sutures infolding the ulcerated area, and, although it is not long since the second operation was performed, the patient appears to be quite free from trouble.

Case 80: At the time of the first operation, which was a gastro-enterostomy for duodenal ulcer, a calculus was felt in the pelvis of the gall-bladder, but owing to the feeble condition of the patient it was thought wiser not to prolong the operation. During convalescence an acute attack of epigastric pain occurred, and similar attacks recurred at intervals for the next two years; these attacks came on soon after food and were more acute than the pain before the first operation, which usually came three hours after a meal. Their character was such that a second operation was decided upon, and the gall-bladder was explored; it was found to be hour-glass in shape and to contain several calculi. The scar of the old duodenal ulcer was seen and infolded; there was no evidence of fresh ulceration. The patient has done well.

Case 100: In this case a recurrence of pain and flatulence, with occasional vomiting and some loss of weight, raised a suspicion of a possible malignant change in the ulcerated area, and the abdomen was reopened sixteen months

after the first operation. There was no evidence of carcinoma, and the pylorus was freely patent. A slight "loop" was present, and so the afferent limb was divided and anastomosed with the efferent by an end-to-side implantation; the pylorus was narrowed by suture. There has been slight improvement in the symptoms as a result.

Case 173: Gave a long history of indigestion with one attack of hæmatemesis at least. Pain varied in time of onset—sometimes immediately, but generally two hours after food. Liquids caused more discomfort than solids, a drink of water producing almost immediate pain. A diagnosis of duodenal ulcer was made, but at operation no lesion could be detected in the duodenum either on inspection or palpation. On the lesser curvature of the stomach, however, slightly nearer the cardia than the pylorus, was a large ulcer with induration extending into both anterior and posterior walls. This was excised and the incision sutured. No gastro-enterostomy was performed. Relief followed the operation for four months, and then pain recurred, coming two or three hours after food and always relieved by the next meal. There was no vomiting. The weight which had been regained was rapidly lost. Fourteen months after the first operation the abdomen was reopened; the scar of the previous excision was found to be perfect, there was no narrowing, and only a few thin adhesions. The first part of the duodenum was surrounded by adhesions, and a large indurated ulcer was found on its anterior aspect. Posterior gastro-enterostomy was performed and the ulcer infolded. The patient has been perfectly well since. There is no doubt that the chief symptoms before the first operation were caused by the gastric ulcer, but the variable time of onset of pain for a few months before operation make it probable that the duodenum was the site of early ulceration even at that time, although no lesion could be detected on examination of the exterior of the gut.

In three cases anterior gastro-enterostomy had to be performed in place of the posterior operation on account of technical difficulties. In two of these (Cases 17 and 169) the loop was divided and the afferent limb implanted into the efferent; and in the third (Case 159) a lateral anastomosis was performed between the limbs.

In six cases gall-stones were removed simultaneously with the performance of gastro-enterostomy. In Case 12 the patient had suffered from repeated attacks of severe colicky pain in the right hypochondrium, accompanied by vomiting and always followed by profuse tarry stools, but no jaundice. In Cases 78, 130, and 161 gall-stones were not suspected before operation.

Case 83 was operated upon for the relief of attacks of typical biliary colic and the gall-bladder drained after the removal of a number of calculi; it is a pity that the gall-bladder was not extirpated, for the patient died less than three years after from carcinoma.

In Case 163 gall-stones were apparently the chief trouble, and a cholecysto-duodenal fistula existed. Case 180 is of the greatest clinical interest. The history of the case and the physical examination strongly suggested obstructive jaundice due to carcinoma of the head of the pancreas, and it was not until

84 Moynihan: *Diagnosis and Treatment of Duodenal Ulcer*

the pathologist's report on the chemical examination of the urine and faeces demonstrated that the obstruction involved the common duct above the level of its junction with the pancreatic that operation was strongly urged. An indurated scar in the duodenal wall was found to be compressing the common bile-duct, and a cholecystenterostomy was performed. Unfortunately the junction had to be made with the colon on account of mechanical difficulties, and no doubt the three short attacks of pain and pyrexia with jaundice which the patient has experienced since operation have been due to cholecystitis. With this exception the operation appears to have brought complete relief.

Cholecystoduodenal fistulae were found in two cases (81 and 163), in the former due to duodenal ulceration, in the latter probably due to gall-stones.

In Case 108 an hour-glass contraction of the stomach was present, necessitating the performance of gastroplasty at the same time as gastro-enterostomy.

Excision of a duodenal ulcer without gastro-enterostomy was the operation in Case 183; scarcely a year has elapsed since the operation, but quite a recent report states that the patient is very well.

Operative Results.—Four patients out of the 186 died as the result of the operation (2.15 per cent.).

Case 16: Acute intestinal obstruction on the tenth day.

Case 33: Uræmia on the third day.

Case 99: Perforation of jejunal ulcer on thirteenth day.

Case 112: Acute tuberculosis on fourteenth day.

Four other patients are since dead at varying periods after the operation:—

Case 30: Four years later from cardiac disease; apparently no recurrence of stomach symptoms.

Case 38: At operation a subacute perforation of a large duodenal ulcer was found. For the first ten months after operation she was entirely free from trouble and gained weight, then pain began to recur, at first at long intervals, but gradually becoming more frequent and severe with occasional vomiting. Two and a half years after operation she began to lose weight rapidly, and ascites and marked anæmia developed with inability to retain food. Death ensued from asthenia and exhaustion three years and four months after operation. The cause of death was probably carcinoma, but whether of duodenum or stomach it is impossible to say, as no autopsy was obtained.

Case 55: In this case a suspicion of carcinoma was raised before operation as the stomach contents were only faintly acid, contained no free HCl, and lactic acid was present. At the operation, however, no evidence of carcinoma could be detected, and the scar of an ulcer was present in the duodenum. The patient's doctor reports that he died two years later from "pernicious anæmia," the symptoms of which had only appeared four months before. There were apparently no symptoms pointing to gastric carcinoma.

Case 83: Operation in this case was undertaken for the relief of symptoms of cholelithiasis, and the gall-bladder, which contained many small calculi, was evacuated and drained. The contracted scar of a duodenal ulcer was present,

so gastro-enterostomy was performed. Two years and nine months later he was readmitted to the Nursing Home with a large tumour in the region of the gall-bladder. An exploratory operation revealed extensive carcinoma involving the gall-bladder and infiltrating the liver. The abdomen was closed, and he died some weeks later.

In the remaining 178 cases an attempt has been made to obtain a report of the present condition of the patient by writing to the doctor who sent the case, and in some instances to the patients themselves. In a few cases the patients have been seen personally within the last few months. In eleven cases (4, 15, 32, 52, 73, 81, 98, 129, 140, 165, and 188) attempts to trace the patients have been unsuccessful, and no report is available at a longer period after the operation than a few months. In four cases (7, 14, 42, and 43) a report was obtained in 1905, but none has been available since that time. In Cases 7 and 14 this report was three-and-a-half and two-and-three-quarter years after operation respectively, and they may be classed as undoubted cures; Case 42 was improved, and Case 43, although the report was only twelve months after operation, appeared to be cured. Of the 163 patients concerning whom recent reports have been obtained, 144 may be classed as cured. The remaining nineteen cases are not yet entirely free from trouble, and details of them are appended:—

Case 17: A case of gastro-enterostomy nine months after operation for perforation of a duodenal ulcer. An anterior operation by Roux's method had to be performed owing to the almost universal adhesions. Patient has now occasional epigastric pain, probably due to adhesions, but is otherwise in excellent health. Very much improved.

Case 18: A very good report in 1905 (three-and-a-half years after operation). Last report not quite so favourable, has occasional bilious vomiting, but is evidently much improved.

Case 23: The patient still suffers from distension and flatulence, but is distinctly better for the operation.

Case 24 (see also Case 40): This patient suffered from severe vomiting, which began one week after posterior gastro-enterostomy, and lasted, with short intermissions, until one year later, when the abdomen was reopened. It was then found that almost all the small intestine had herniated through the opening in the transverse mesocolon. This was reduced and a lateral anastomosis between limbs of loop performed. Patient is much better, but still has occasional pain and vomiting.

Case 36: Eighteen months after operation reported to be little better. Recent report not very reliable, but appears to be better than he was.

Case 49: Suffers from attacks of regurgitant vomiting, but says she is much better.

Case 70: Occasionally vomits, but is otherwise very well.

Case 74: Still has attacks of regurgitant vomiting every three or four weeks, which he relieves by lavage, but is at work and otherwise well.

Case 75: Occasional pain and vomiting, but is much improved. A neurotic.

86 Moynihan: *Diagnosis and Treatment of Duodenal Ulcer*

Case 76: Still suffers from flatulence and anæmia, but is much improved.

Case 88: Occasional pain and "water-brash," but much better.

Case 91: Regurgitant vomiting of bile about once a week, causing no pain. Otherwise quite well.

Case 100: Much relieved for one year, then recurrence of pain and distension, with occasional vomiting. Fifteen months after gastro-enterostomy, abdomen opened, as carcinoma was suspected; none found. As a "loop" existed, lateral anastomosis was performed. Recent report (one year after second operation) states that a ventral hernia has developed; he is better, but not well.

Case 104: Much improved; practically cured.

Case 113: Says he is no better, although doctor thinks he is. Is a hypochondriac.

Case 125: Still some pain, but has gained 11 lb. in weight, and is on the whole better.

Case 161: Patient suffers from cardio-spasm, and is still under treatment.

Case 176: Not much improvement six months after operation. Patient is a busy practitioner, and is apt to overwork, but when seen recently was much better.

Case 179: Slow improvement taking place six months after operation; one year after operation much better and back at work.

Of Case 113 one is bound to say that the patient appears to be no better, and in Case 161 improvement is doubtful; the other seventeen cases are distinctly benefited, if not cured.

Summarizing, therefore, the results of our inquiries, we have the following results: 163 recent reports—cured, 144; improved, 17; doubtful improvement, 1; no better, 1. Four reports in 1905: cured, 3; improved, 1. Cases not traced, 11. The operative results work out as follows:—

Died as result of operation	...	4	...	2.15 per cent.
Died at varying periods of other causes than operation	...	4	...	2.15 "
Cured	...	147	...	79.0 "
Improved	...	18	...	9.6 "
Doubtful improvement	...	1	...	0.5 "
No better	...	1	...	0.5 "
Not traced	...	11	...	— "
		186		

Cases in which Post-operative Vomiting Occurred.—In the following twelve cases vomiting occurred for a variable length of time after operation, or is still present:—

- Case 6.—Now cured.
- " 18.—Occasional bilious vomiting still present.
- " 24.—Severe vomiting due to hernia into lesser sac, much relieved. (See also Case 40.)
- " 27.—Temporary regurgitation. Now cured.
- " 34.—Temporary regurgitation. Now cured.
- " 42.—Occasional vomiting one year after operation. No later report.
- " 49.—Regurgitant vomiting still persists.
- " 70.—Still vomits occasionally.
- " 74.—A typical attack of regurgitant vomiting every three or four weeks.
- " 91.—Regurgitant vomiting still persists.
- " 100.—Occasional vomiting. Reoperated with some relief.
- " 104.—Occasional bilious vomiting.

Of these cases of post-operative vomiting, only six (24, 27, 34, 49, 74 and 91) can be classed as true regurgitant vomiting. In Case 24 the vomiting was severe, and the cause removed at a second operation. In Cases 27 and 34 the vomiting was temporary, and the cause is not known, unless it be that a loop was left between the anastomosis and the flexure. In Cases 49 and 91 the stomach was found to be very much dilated, and there may be some mechanical difficulty, which still causes the vomiting to persist. In Case 74 the operation was performed by the antiperistaltic method.

NOTE.—Up to December 1, 1909, operation has been performed in 228 cases, with four operative deaths. The last fatal case was in June, 1907 (Case 112). In the last 116 consecutive cases there have been no deaths.

Sir LAUDER BRUNTON, F.R.S., said he was greatly honoured by the request that he should open the medical side of the discussion on the very admirable paper which had been presented. But in view of the number of prospective speakers he would limit his remarks as much as possible. With regard to the season, it had been his experience that gastric and duodenal ulcer—and he could not distinguish the two conditions in his list of cases—were much more common in two periods of the year than at other times. They seemed much more common in autumn and spring, and he thought the reason was that people took to their winter clothing too late, and put off that clothing too soon in the spring, the result at both periods being probably chills, and chill was one of the sources of such ulcers. Years ago, when he was working at the physiology of the subject for Burdon Sanderson's "Handbook for the Physiological Laboratory," he made many experiments on the stomach and intestine, but his attempts to produce gastric ulcer were futile. He put a ligature around a small portion of the mucous membrane of the stomach, which he had previously opened. He then closed the wound, and, keeping the animal narcotized, he allowed it to remain four or five hours. But in none of the cases did an ulcer form. He might supplement one point which Mr. Moynihan left out—namely, in reference to pain. Pain came on two to four hours after a meal, because the stomach then commenced to discharge its acid contents into the duodenum, and these, passing over the surface of the ulcer, caused intense smarting. That pain could be relieved in two ways: (1) by the patient putting anything into the stomach which caused the pyloric orifice to contract. Thus the acid juice from the stomach was prevented from flowing into the duodenum, and any acid present in it was readily neutralized by the alkaline juices of the duodenum itself, and possibly also by the bile. (2) That process could be imitated by giving large doses

of bicarbonate of soda. Sometimes one might be misled by the nature of the lesion and yet be correct as to its position. In one case brought to him the symptoms were pain four hours after food, and he concluded there must be something wrong in the duodenum, but it did not yield in the ordinary way to alkali. He did not see the case again, but eighteen months later the *Lancet* contained a description of an operation on that patient. It showed the lesion in the duodenum to be a kink, not an ulcer. Time had been an important aid to him in diagnosis. He was once asked to see a patient who was suffering from very severe lumbago. He found the so-called lumbago limited to the right loin, and the pain came on between two and three hours after a meal, and was again stopped by taking food. He concluded it was duodenal ulcer, and said that could be tested by giving large doses of bicarbonate of soda, which would stop the pain by neutralizing the acidity of the gastric contents. That was given, and the pain ceased immediately. Two or three days later the patient passed a large quantity of blood in the stools. He found the patient had been allowed to get up, and he ordered him to bed, and ordered something to be added to the bicarbonate of soda, as he remembered that that might make the surface of the ulcer softer. So carbonate of lime was put in, to act as an astringent and styptic. But it also tended to constipate, so that now his practice was to add also carbonate of magnesia to counteract this effect of lime. One should always employ this test in addition to paying attention to the history. He never considered ulcer to be present unless the pain was stopped by the combination he had mentioned. In one case that means was not successful, and he wondered at it. The pain was not on the right side, but rather under the left ribs, and that fact further misled him. He put the patient to bed and kept her on rectal feeding three or four days, and then gave a very limited diet. At the end of that time she began to improve, but still had some recurrences of pain. She had suffered for about two years from the pain after meals, lasting a short time and then disappearing. She had had no jaundice, and there was no tenderness over the liver. After being in hospital for a time she had what seemed to be perforation; she was collapsed, and lay perfectly still on the bed. In all other cases of gall-stone he had seen, the patient writhed about in agony. He asked a surgical colleague to see her, but he said he could not operate then as she was too collapsed and would die under it. She recovered, and when operated upon no ulcer was found; but there were between 200 and 300 minute gall-stones of the size of a millet seed, each of which was sufficient to scrape through the

cystic duct without blocking the common bile duct. There were so many that she evidently had had a sufficient supply to last her two years, one for each meal, and there were enough left to serve for another 100 days in the same way. That showed that the diagnosis of ulcer from gall-stones was not always easy. He wished to insist on (1) the necessity of keeping the surface warm, so as to prevent the occurrence of either gastric or duodenal ulcer; and (2) on the use of mixed carbonates in large doses as a means of diagnosis. Those carbonates would also relieve the pain in patients who would not agree to operation; and if they were combined with warmth and careful dieting, surgical measures in many cases might be avoided. But he agreed that where an ulcer was obstinate, and did not yield to treatment, operation should be urged.

Dr. W. HALE WHITE said he thought one thing which must interest the members present was not so much the part of the subject which had been spoken on as that dealing with the frequency with which cases of duodenal ulcer were encountered in ordinary medical practice. Therefore he got out a few figures on that point. The statistics of Perry and Shaw, as all who had gone into the literature of the subject knew, showed that out of 17,000 post-mortems at Guy's Hospital there were only 70 cases of duodenal ulcer. That went down to the year 1892. But in the 3,800 post-mortems for the last seven years there had been 38, so that seemed to show an enormous increase in the number of ulcers found. But on looking into the cases it was seen that the reason of that increase was that now the medical profession generally recognized that the patients' only chance, when perforation of gastric or duodenal ulcer occurred, was to send them to a hospital as quickly as possible; whereas in the old days trouble was not even taken to send them to hospital, so necessarily fatal were they considered. Again, looking at the matter from the same statistical point of view, in the last 20,000 medical cases admitted to the hospital duodenal ulcer was diagnosed 33 times. But taking the last 1,000 cases seen outside the hospital, duodenal ulcer was diagnosed 24 times, showing apparently a much greater proportion of duodenal ulcers among private patients outside the hospital than in those seen inside it. But there, again, the fallacies were many, because in looking into the cases one saw that of the 33 in hospital, 17 were fatal; there were many perforative cases. And of the 24 outside the hospital only one was fatal, and that fatality was due to hæmorrhage. That comparison showed there must be many people suffering from duodenal ulcer whose symptoms were not considered severe enough to secure their admission to hospital; and some of these who lived near hospitals came in, late in

their illness, for the fatal effects of the ulcer—perforation or hæmorrhage. So it must be concluded that for a large number of people with duodenal ulcer, hospital out-patient treatment sufficed. Therefore probably the number of cases of duodenal ulcer was much greater than the figures he had quoted showed, for, with regard to the private cases seen outside the hospital, in all the patient was sufficiently bad for a second opinion to be sought. So that the apparent greater frequency of duodenal ulcer, as compared with twenty years ago, was due to the greater appreciation of the condition, for which the profession had largely to thank Mr. Moynihan in this country and the Mayo brothers in America. With regard to the symptoms, he had thought over what seemed to him a picture of the pain of duodenal ulcer, but he would not weary the meeting with it, as it practically confirmed what Mr. Moynihan had said. The strange part which misled many people was the periodicity of the pain. The patients would go for months without pain. He had known sufferers from duodenal ulcer who could go out to dinner several nights a week for two or three months, and then have an attack which was so bad that he had seen such a patient writhing on the floor with the pain, and that man was proved by operation to have a duodenal ulcer.

One thing he would add to Mr. Moynihan's description, and which he (Dr. Hale White) was accustomed to lay much stress on, was local tenderness. Many cases showed local tenderness in the neighbourhood of the duodenum. An important point was a slightly greater rigidity of the right rectus than the left. But it was this very local tenderness which led to what he thought was the greatest difficulty in diagnosis, for he agreed with Mr. Moynihan that the diagnosis of duodenal ulcer was not difficult. Of the recent cases he had looked up he found that when surgery was carried out the diagnosis was proved to have been correct in every case except one. But he had sometimes found it difficult to determine whether a case was duodenal ulcer or gall-stones, not when patients were passing gall-stones, but when they were suffering pain due to gall-stones lying in the gall-bladder, too large to pass down. If there had been time he could have mentioned a case which was confidently thought to be due to duodenal ulcer. At the operation, as far as could be seen outside, the duodenum was healthy, but the patient had a large stone in the gall-bladder, which was removed, and the pain had now ceased. Owing to the close contiguity of the two parts, possibly the symptoms were due to some trouble in the duodenum proceeding from the gall-bladder. He had come across one or two interesting complica-

tions which did not seem to have occurred in Mr. Moynihan's series. One patient had considerable parotitis; and within the last three months he had seen two subphrenic abscesses in connexion with duodenal ulcer, one yielding nearly 2 pints of pus. Subphrenic abscess due to duodenal ulcer must almost necessarily be to the right of the suspensory ligament; and most subphrenic abscesses to the right of that ligament were due either to duodenal ulcer or to the appendix. He thought the appendix cases never contained air, but duodenal ulcer cases might. In one case, because the abscess cavity contained air, it was concluded that it was caused by a duodenal ulcer, and that proved correct. Another complication he had come across was that once, in a bad case of hæmorrhage, he could feel a large blood-clot completely filling the duodenum. The hæmorrhage came on while the patient was lying in the hospital, and it proved fatal. The ulcer was found at the post-mortem. Mr. Moynihan had carefully said that operation was to be done for chronic duodenal ulcer. That, of course, presupposed that there should be a stage in the illness during which the patient should be allowed the benefit of other treatment. The more or less routine treatment he had adopted was to put the patient rigidly to bed, and he was fed on a mixture of one egg and $\frac{1}{2}$ pint of milk, taking a little at a time every half-hour. The quantity was gradually increased, until 2 or 3 pints daily were being taken. Cream could be added to get in the fat which was so useful; or olive oil could be given, and the additional advantage of that was that it was slightly aperient, for these patients were often constipated. After a month of that treatment they were given chopped chicken, &c. If, after such treatment, the pain recurred, he would certainly hand over the case to a surgeon. For one severe or several less severe hæmorrhages it was probably wise to do short-circuiting, even without waiting for medical treatment, for there were many fatalities from hæmorrhages due to duodenal ulcer. He would like to ask Mr. Moynihan whether it was not an objection to excising an ulcer that the ulcers were often multiple, in which case often although one ulcer had been excised, others would be left.

Mr. HERBERT WATERHOUSE said he was glad to find himself in almost complete agreement with Mr. Moynihan. That was not surprising, as English surgeons had learned most of what they knew about duodenal ulcer from Mr. Moynihan. One point which had struck him much in duodenal, as compared with gastric, ulcer was the comparative frequency of the two in hospital and private practice. In hospital practice he had had a much larger number of cases of gastric than of duodenal

ulcer; whereas in private there had been three or four duodenal to one gastric. He attributed the difference largely to the frequency with which gastric ulcer occurred in young anæmic servant-girls. The duodenal ulcers which he had met with on the operating table had proved to be indurated, chronic, and always easily felt and demonstrable. He agreed that an ulcer which could not be felt by the surgeon or his assistant—he would not say which could not be demonstrated to the onlookers in the operating theatre—was not to be called a duodenal ulcer. As to age, his statistics were in striking agreement with those of Mr. Moynihan. Probably nine-tenths of the cases he had come across ranged from 30 to 45 years of age. Duodenal ulcer was not unknown in childhood. He had had one case in a child under a year old, and another under 5 years of age. Writers on the subject appeared to have overlooked the possibility of duodenal ulcer occurring in childhood. Mr. Moynihan had done good service by pointing out that duodenal ulcers were often multiple. He (the speaker) had never found more than four in the living patient or on the post-mortem table. He thought the ulcers were multiple in about one case in three or four; and that fact should be a bar to any such operation as excision. He used, and would continue to use, for this condition only one operation—viz., gastro-enterostomy. In spite of what Mr. Moynihan had said, he regarded excision of the ulcer as dangerous, and it must have a greater mortality than gastro-enterostomy, since in this operation the mortality, in careful hands, was practically *nil*. He would like one result of the debate to be a general agreement that excision of duodenal ulcer was to be a thing of the past; that every case could be treated satisfactorily, sufficiently, and with the maximum of safety by gastro-enterostomy. He referred to the frequency with which he had found gastric and duodenal ulcers in combination, perhaps in one case out of every four. That led him to speak of the difficulty of diagnosis. Mr. Moynihan and Dr. Hale White said that the diagnosis of duodenal ulcer was not usually difficult. It might not be to those gentlemen, but to ordinary surgeons like himself he feared there would often be difficulty in diagnosis. Usually where he had been uncertain whether there was gastric or duodenal ulcer he had found both those conditions. He therefore did not wish it to be understood that all in the meeting agreed that the diagnosis of duodenal ulcer presented small difficulty. He had known mistakes made in diagnosis where it was a question of duodenal ulcer versus gall-stones. He had seen cases of duodenal ulcer in which the pain radiated in a marked manner. As a rule the

pain of duodenal ulcer was localized and did not radiate, and that was usually a diagnostic point between those ulcers and gall-stones. Duodenal ulcers sometimes caused pain which could be well described as agonizing; and in such cases it was difficult to distinguish between ulcer and gall-stones. Again, those two conditions were not infrequently found in combination. He agreed with Dr. Hale White as to localized tenderness and slight rigidity of the right rectus being regarded as valuable diagnostic signs. He agreed with Mr. Moynihan as to the absence of hyperacidity. In all the cases which he had had examined, the free hydrochloric acid had been under, rather than above, the normal. An important point in the diagnosis was melæna. That had been present in most of the cases which he had had to deal with; and where no melæna had been obvious, the microscopical examination of the fæces had, in a fairly considerable proportion, revealed the presence of blood. It was a source of gratification to surgeons to know that the two distinguished physicians who had already spoken realized thoroughly that when medical treatment had failed it should not be prolonged to any great extent, but the case should be handed over to the surgeon before any harmful results—such as stenosis—had had time to develop. He knew of no operation which gave greater relief in any surgical condition than did gastro-enterostomy for duodenal ulcer.

Mr. FREDERIC EVE said: In my experience the symptoms of duodenal ulcer are fairly definite. Nevertheless, we should be on our guard against assuming that a group of symptoms is characteristic of this condition. They may certainly, in my opinion, be perfectly simulated by a functional disorder of the stomach (call it hyperchlorhydria or what you like) without any organic lesion. Although I have myself operated on few cases of duodenal ulcer in comparison with gastric, yet until a fortnight ago I had always found an ulcer when its presence was suspected. Bearing on the question of diagnosis, the following case seems worth quoting:—

Suspected Duodenal Ulcer.—W. O., aged 30, Salvation Army officer, had suffered with periodic attacks of abdominal pain for nine years altogether. A year ago he had an attack that lasted six or eight months. His present attack commenced twelve weeks before his admission to the hospital on November 11, 1909. He complained of pain in the epigastrium coming on two to three hours after a meal. He often awoke in the night with an attack of pain which was relieved by taking warm milk; this fact was verified by the sister and nurses.

He had no vomiting, hæmatemesis, or melæna. He complained of tenderness on deep pressure over the region of the duodenum. On the occasion of the second examination a very faint trace of blood was found in the stools, but the importance of this was rendered doubtful, as he had suffered with hæmorrhoids. Examination of a test-meal gave total acidity 96 per cent., free hydrochloric acid 0·27; hence there was distinct hyperchlorhydria. At the operation the duodenum was exposed, and on palpation I could find nothing abnormal. A colleague who was assisting me noticed a thickening with some wrinkling of the peritoneum over a small area of the anterior surface of the first part of the duodenum, which he suggested might indicate the position of an ulcer. I could, however, feel no thickening beneath it. An incision was made on the anterior surface of the stomach near the pylorus and the finger passed into the duodenum. Nothing was found by myself or by my colleague, who also introduced his finger. No ulcer of the stomach was felt by palpation either externally or by the finger within the viscus. Gastro-enterostomy was not performed. This case, in which there had been repeated attacks of pain indistinguishable from those usually associated with chronic duodenal ulcer and in which definite "hunger pains" were present, shows that this combination of symptoms is by no means characteristic of chronic duodenal ulcer. The attacks showed distinct periodicity and had extended over a long period. From the first I was doubtful of the case, and therefore kept him in my wards for observation for three weeks. He was allowed to get up, and latterly was given ordinary diet to see if traces of blood could be found in the fæces. The "hunger" pains were well marked, as I satisfied myself by questioning the sister and nurses.

Gastric ulcer, even when situated near the cardia, may also simulate duodenal ulcer. In the following case, from the wards of my colleague, Dr. Percy Kidd, duodenal ulcer was diagnosed by several good observers:—

T. B., aged 36, was admitted under Dr. Percy Kidd on October 25, and transferred to my care after he had been a month under medical treatment. He had been suffering from indigestion on and off for several years; had been ill for six weeks before admission, with pain coming on two to three hours after food. He vomited twice during this period. The pain was referred to the lower part of the abdomen, and was dull and aching; but at times came on suddenly and doubled him up. No tenderness or pain over the duodenum. He awoke at night with pain, which was relieved by taking hot milk. There was distinct

tenderness on deep pressure in epigastrium to the left of the ensiform cartilage. At the operation on December 6 a chronic ulcer was found on the lesser curvature about 2 in. from the cardiac orifice. The duodenum was perfectly healthy.

There can be no doubt, in my opinion, that the diagnosis of duodenal ulcer is often beset with great difficulties, and its onset is very insidious. For example, a patient who died in the London Hospital from perforation had only complained of pain—one and a half to two hours after meals—for one week. Two chronic ulcers were found in the duodenum, on the anterior and posterior walls respectively. If it were necessary to enforce the importance of this subject, I might appeal to the mortality from duodenal ulcer at the London Hospital, as shown by the post-mortem reports of 1907 and 1908. During this period there were 27 cases, of which 21 died of perforation and one from erosion of an artery. In the remaining 5 cases the ulcer was secondary to other affections. Nineteen of the cases were males and 8 females, and the extremes of age were 7 weeks and 71 years. Two patients died of perforated duodenal ulcer at the ages of 65 and 71 years respectively. The ulcers which perforated were chiefly on the anterior wall, evidently for reasons similar to those existing in the case of the stomach; and in 7 of these cases of primary duodenal ulcer—namely, a third—there was a “contact” ulcer on the opposite surface of the duodenum. In 5 of these cases an ulcer co-existed in the stomach. Dr. Turnbull informs me that he does not remember to have met with any instances of cicatrices due to healing of ulcers in the duodenum. As regards the five cases of ulcer of the duodenum which were secondary to other conditions, it may be of interest to mention that one was associated with severe sepsis due to gangrene of the leg, another with anæmia and malignant disease of the cervix uteri, and the third was due to hydrochloric-acid poisoning. Two instances occurred in infants: one was associated with general tuberculous ulceration of the intestine, and the other with multiple ulceration along the intestinal tract.

I fully concur in the opinions expressed—that suggested cases of duodenal ulcer should be operated on if they do not yield to medical treatment, or on a second attack of pain occurring.

My experience of the operation of gastro-enterostomy has been favourable and the results permanent, except one patient, a male, aged 35, who died of uncontrollable melæna forty days after the operation and ten days after returning from a convalescent home. This operation therefore cannot be considered as absolutely curative in every case. Owing to this case, I determined in future to constrict the duodenum

by gathering sutures in order to insure the permanent patency of the gastro-enterostomy opening.

I consider Mr. Moynihan's plan of infolding an ulcer when situated on the anterior wall a good one, especially in view of the fact that in many cases another ulcer co-exists on the opposite wall. It does not seem to me either necessary or advisable to excise the ulcer, and this view is again supported by the frequency of the existence of "contact" ulcers, which could only be removed by resection of a portion of the duodenum. It has not been my invariable experience that the ulcer is plainly seen and demonstrated, for in two instances I found it necessary to explore the duodenum by an incision in the stomach in order to be sure that an ulcer existed. In one of these cases the head of the pancreas was indurated and the duodenum was adherent to it, but no change was discernible in the remaining portion of the circumference of the bowel. On passing my finger through the pylorus by means of an incision in the anterior wall of the stomach, an ulcer of small circumference was found on the inner wall of the duodenum, which deeply penetrated the pancreas. This was the patient who subsequently died of hæmorrhage. The incision was used for the purpose of anterior gastro-enterostomy, the posterior wall of the stomach being firmly adherent to the parietes. In the other patient gastric ulcer was suspected, and he had hæmatemesis and no melæna. No ulcer being found on external examination of the stomach, I examined its interior with negative results, but found an ulcer of the duodenum on passing my finger through the pylorus.

Too much stress should not be laid on superficial changes and thickenings of the peritoneum. In examination of the surface of the stomach for ulcer, I have found changes in the peritoneum strongly suggestive of ulcer beneath, which subsequently proved on examination of the interior of the stomach with the speculum and finger to be illusory; and I have quoted a similar case in regard to the duodenum.

For the sake of scientific accuracy I commend the method of examination of the interior of the stomach and duodenum in doubtful cases to my surgical colleagues, for I venture to think it has thrown important light on the pathology of gastric hæmorrhage. In 29 consecutive cases diagnosed as gastric ulcer, in 21 the interior of the stomach was examined. In 12 of these no ulcer was found, and the symptoms were due to gastrotaxis or allied conditions. In one the ulcer was in the duodenum. My experiences in this relation were reported in the *Lancet*.¹

¹ 1908, i, p. 1822.

Surgical Section.

January 11, 1910.

Mr. G. H. MAKINS, C.B., Vice-President of the Section, in the Chair.

Discussion on "The Diagnosis and Treatment of Duodenal Ulcer."¹

Dr. ROBERT HUTCHISON said that the chief interest to the physician in the discussion which was reopened that day resided in the question of diagnosis. Speaking for himself, if he were convinced that duodenal ulcer were present he would have no hesitation in recommending surgical treatment. He did not propose, in the course of his remarks, to consider the question of treatment at all; as to the precise form of surgical treatment to be adopted, the physician had no right to offer an opinion.

Mr. Moynihan had pointed out that physical signs could not be depended on in the diagnosis of this condition, and that the history given by the patient was of greater assistance. In particular he (Mr. Moynihan) emphasized the importance of "hunger-pain" as a symptom. So far as the discussion had gone, however, it had tended somewhat to impair the diagnostic value of hunger-pain. He did not say that Mr. Moynihan over-emphasized this point in opening this discussion, but certainly a superficial student of his writings might be excused if he gathered that Mr. Moynihan regarded hunger-pain as a pathognomic sign of duodenal ulcer. Now, the discussion had shown that the presence of stone in the gall-bladder might give a pain which could not be distinguished from the hunger-pain of duodenal ulcer. Chronic appendix trouble might also give a hunger-pain. Here, therefore, were two conditions which tended to impair the diagnostic value of

¹ Adjourned from December 14, 1909.

hunger-pain in deciding whether a patient had duodenal ulcer or not. Both of these conditions, however, were organic in nature, and no matter whether a patient had gall-stone, chronic appendix trouble, or duodenal ulcer, he was a fit subject for operation. But what concerned physicians more particularly was this: Was there a condition, not organic at all, but functional, which manifested itself by the presence of a pain in the late period of digestion? He believed that there was such a condition—namely, that to which the term “hyperchlorhydria” was commonly applied. He was not concerned to define the term “hyperchlorhydria.” The important question was as to whether such a condition existed at all, and whether it was associated with hunger-pain as one of its chief symptoms. No one, he thought, would be sceptical as to the existence of hyperchlorhydria. Large collections of cases showed that it did occur, cases in which the excess of acid was demonstrated by the giving of a test meal. He believed that such patients did have as a symptom a pain which could not be described as other than hunger-pain. He took it that Mr. Moynihan would reply that these were not cases of hyperchlorhydria merely, but that there was present in all of them a duodenal ulcer. And that was the point at issue between the physician and the surgeon in this matter. It was not easy to prove the existence of a functional disease, seeing that the physician did not perform operations and that patients did not die of functional disorders; nevertheless, the physician had perhaps less difficulty in conceiving a functional condition than the surgeon, who was more apt to be biased in favour of organic conditions. Patients had, at all events, suffered from hyperchlorhydria, and, on their death from another cause, careful examination had discovered no organic lesion. He concluded, therefore, that hyperchlorhydria might exist for many years purely as a functional condition, and it was his (Dr. Hutchison's) contention that this functional condition was associated with hunger-pain as a symptom. If they were asked to prove the absence of an ulcer in cases of hunger-pain, only two lines of evidence were possible. One of these was the result of operation in early cases, and the other possibly post-mortem evidence. There had not been many operations after merely one attack of hunger-pain. In one such case Mr. Moynihan admitted that it took a certain amount of pathological ingenuity to demonstrate the ulcer at all, but it was just possible to get a pathologist to say that he thought there had been an ulcer there. He had heard of another case in which no ulcer was found after one attack of pain, and if there was only one case showing no ulcer after an attack of hunger-pain

it gave the whole matter away. If it were not possible to find any organic lesion in even one case after definite hunger-pain, it might be safely concluded that the condition was due to hyperchlorhydria alone.

As to post-mortem evidence, every physician would admit that in young men and men in early middle life hunger-pain was a relatively common symptom. If that were so, and if they admitted for the sake of argument that hunger-pain always meant duodenal ulcer, then he thought everyone would say that there ought to be a great deal more post-mortem evidence of scars in the duodenum than there was. The only escape from that dilemma was to contend that the ulcer might leave no sign of its presence afterwards. He did not think that Mr. Moynihan would maintain that. If, therefore, ulcer always left a scar behind it, and if hunger-pain always meant duodenal ulcer, these scars ought to be common. But out of some 2,000 post-mortem examinations at the London Hospital, the patients all having died from some other condition, indications of old duodenal ulcer were only found in three cases. In patients who had had phthisis early in life which had healed up, and who had subsequently died from something else, scars in the lungs were found. Why, therefore, should not scars be found in patients who had had duodenal ulcer? He thought there was a danger of losing one's perspective when one was dealing to a certain extent with selected material. Was it not possible that Mr. Moynihan regarded duodenal ulcer as more common than it really was because he had such a large number of cases coming into his hands for treatment? Any form of specialism tended to make one lose perspective. The alienist, for example, thought we were all more or less mad. Anyone who saw a large number of patients of one class did unconsciously get hold of the idea that the particular condition was commoner than it really was. But surgeons might fairly ask, What was the relation of hyperchlorhydria to duodenal ulcer? Was there any relation at all? His own idea was that a condition of congestion, hyperæsthesia and hyperacidity of the stomach might last for a considerable time, with intervals between the attacks, and it was perfectly easy to understand that such a condition predisposed to the formation of ulcer. It might be that after this condition had existed for some length of time an ulcer formed. The difficulty would be to say definitely when a patient had passed the border-line—when he had ceased to have the condition of congestion and hyperacidity alone and when he had gone on to the formation of an ulcer. Of course, given such a condition as hæmorrhage, the matter was settled once for all. But everyone knew that there might be duodenal

ulcer long before there was melæna. That raised a practical question which it was very desirable to settle in the course of that discussion: At what time should one operate? Mr. Moynihan had admitted that in the early stages of duodenal ulcer, or in what he (Dr. Hutchison) regarded as the stage of "hyperchlorhydria," the case was one for the physician. He wished to ask Mr. Moynihan how many definite attacks of hunger-pain should take place before it was right to operate. That was one of the most important questions for the physician to settle. His final point was this: After these cases had been operated upon, granting that a duodenal ulcer had been present, he still thought the patients should be kept under medical treatment for a considerable time. The patient should not be allowed to have an unrestricted diet after the operation was over. The operation was not the end of the treatment, and the patient still needed to be kept under the watchful eye of the physician.

Dr. A. F. HERTZ: Until eight years ago it was universally believed, as a result of the study of the clinical history of patients who were found after death to have a duodenal ulcer, that duodenal ulcer was a rare condition, occurring with one-twentieth the frequency of gastric ulcer, and that its diagnosis was generally impossible before hæmorrhage or perforation had led to a fatal issue. Being brought up in this belief, I could not but read with scepticism Mr. Moynihan's earlier publications on duodenal ulcer, which suggested that it was a comparatively common disease, which could be diagnosed with certainty even in the absence of hæmorrhage. I felt, however, that one could learn more on the subject from a surgeon who had operated on over 200 cases than by studying post-mortem records or by chemically investigating the contents of stomachs the anatomical condition of which was a matter of speculation. I was therefore very glad to avail myself on three occasions of Mr. Moynihan's kind offer to allow me to examine his patients, and subsequently, at the operation, to see him demonstrate the condition present. From my experience in Leeds I cannot help agreeing with Professor Osler's dictum that "we physicians have been napping, and that what the modern gastro-enterologist needs is a prolonged course of study at such surgical clinics as Leeds or Rochester, Minnesota."

I am convinced that we must reject the old teaching based on pathological observations, and accept, with certain reservations, Mr. Moynihan's conclusions as to the frequency and the symptoms of

duodenal ulcer. The condition is certainly far more common than was formerly believed, but not, I think, as common as the 200 cases operated upon by a single surgeon in eight years would lead us to expect. When a surgeon has a reputation for the treatment of a particular condition, patients naturally come from all parts of the country to be operated upon by him, and I may remark, in this connexion, that not one of the ten patients I saw operated upon by Mr. Moynihan lived in Leeds.

There can be no doubt that vast numbers of people with duodenal ulcer have been cured without anybody having suspected that they were suffering from such a serious condition, their symptoms having been ascribed to various functional disorders, such as nervous dyspepsia and hyperchlorhydria. But that does not mean that nervous dyspepsia and hyperchlorhydria do not exist. The former, at any rate, is infinitely more common than duodenal ulcer, but, for want of accurate knowledge of the symptoms of duodenal ulcer, examples of this condition must have constantly been incorrectly grouped with others of a less serious nature. There is more excuse for the confusion with functional hyperchlorhydria than one would gather from Mr. Moynihan's statement that the amount of hydrochloric acid in his series of cases was not increased. Though that may be the case in the West Riding of Yorkshire, it seems to be established beyond doubt that in most parts of Great Britain duodenal ulcer is almost always associated with hyperchlorhydria. These conflicting results are doubtless due to local differences in diet, corresponding to those which explain the fact that hyperchlorhydria is almost constant in patients with gastric ulcer in North Germany, but comparatively rare in South Germany. There can, moreover, be no doubt that the presence of free hydrochloric acid—whether excessive or not—plays an important part in the production of the pain of duodenal ulcer, as the pain is immediately relieved by alkalies and by the removal of the acid fluid present in the stomach. I cannot, however, agree with Sir Lauder Brunton that the hydrochloric acid itself is the direct cause of pain, as I have found that several ounces of 0.5 per cent. hydrochloric acid produce no pain when introduced by a tube into the empty stomach of patients, in whom an operation performed the same or the following day revealed the presence of an active gastric or duodenal ulcer.

The occurrence of discomfort or pain in the early part of the night is an important symptom. Two of my patients consulted me more on account of the insomnia caused by the feeling of epigastric discomfort,

which kept them awake for an hour or two every night, than on account of the hunger-pain they experienced during the day. It is noteworthy that the interval between the evening meal and the onset of the pain, which wakes the patient in the night, is always an hour or two longer than the interval between the other meals and the pain to which they give rise. This is due to the evening meal being generally the largest, and to the fact that the activity of peristalsis is diminished during sleep, with the result that the evacuation of the stomach is retarded.

The long interval between a meal and the onset of pain has generally been ascribed to reflex spasm of the pylorus, causing delay in the passage of food from the stomach. But I have found, by X-ray observations on three of Mr. Moynihan's and four of my own cases of duodenal ulcer, that there is generally no delay in the emptying of the stomach, which always commences immediately after food is taken. The pain begins when the stomach has evacuated about three-quarters of its contents, and its spontaneous disappearance after about an hour seems to correspond with the moment when the stomach becomes completely empty.

If the patient, when first seen, is already on a strict diet, the diagnosis is often very difficult: the exact relationship between the taking of food and the onset and relief of pain is then only obtainable by putting the patient for a short time on to a full diet. Mr. Moynihan places no reliance on the results of physical examination, but I must agree with Dr. Hale White as to the importance of tenderness over the painful area and rigidity of the right rectus compared with the left, although the pain may be localized exactly in the middle line or even in the left side. These signs, though occasionally absent throughout, are most marked and sometimes alone present, if the patient is examined about three hours after a meal, when he is actually in pain.

I had the good fortune to see the man upon whom Mr. Moynihan operated during a first attack. He had been ill for nine months, and had had most typical hunger-pain after almost every meal during all that time, but at the operation no sign of a duodenal ulcer was found. The small fibrous scar described by the pathologist was invisible to the naked eye, and could hardly have caused the symptoms, which were present in their full intensity up to the very day of the operation. Mr. Sherren tells me that he also, at the earnest request of a patient, has operated during a first attack—and found nothing. It is clear, therefore, that a single attack of hunger-pain, even if it lasts for as long as nine months, does not necessarily mean duodenal ulcer, though perhaps it indicates

a condition which will lead to ulceration in the absence of suitable treatment.

Mr. Moynihan lays great stress on the significance of the recurrence of attacks, between which the patient is entirely free from pain, often for several months at a time. This is, I believe, a result of the remarkable ease with which duodenal ulcers heal, as it is scarcely possible that an open ulcer is present in the intervals between the attacks. Many of Mr. Moynihan's cases are operated on in the quiescent period, when hunger-pain is no longer present. I think that the white scar seen on the outer surface of the duodenum in such cases must be simply the remnant of an ulcer which has healed. As the inside of the duodenum is hardly ever inspected, it is impossible to say with certainty whether this view is correct, but I have at autopsies seen precisely similar scars on the outer surface of the duodenum and the stomach, the mucous membrane of which was absolutely healthy.

Vermehren has recently reported a case in which he discovered a thick whitish scar on the front of the pylorus in a woman who had suffered from symptoms of gastric ulcer. A gastro-enterostomy was performed, but the patient died two days later from broncho-pneumonia; at the autopsy no trace of ulceration was found in the mucous membrane of the stomach or duodenum.

The white scar on the under surface of the duodenum, seen at the operation in many cases, is in striking contrast to the appearances which, I imagine, must indicate the presence of active ulceration. I was fortunate to see Mr. Moynihan operate on a medical man, aged 30, who was having very severe hunger-pain up to the time the operation was performed. The external examination of the duodenum showed the presence of a thick red mass which was clearly the outside of an active ulcer, but there were three small white scars in addition. As the patient had had attacks of hunger-pain for the last twenty years, it seems highly probable that the earlier attacks were due to the presence of ulcers, which were now healed and were represented by the white scars, but that the recent pain was due to the active ulcer.

According to Mr. Moynihan, the treatment of chronic duodenal ulcer should always be surgical. To my great surprise, Dr. Hutchison agrees with this opinion. I am, however, convinced that this is erroneous. I have already given reasons for believing that the scars found at operations in the quiescent period represent healed ulcers, and every practitioner must have seen many people with the symptoms, which we now believe to indicate the presence of a duodenal ulcer or a pre-ulcerative

condition, get well and remain well with medical treatment. If it were true that nothing but surgical treatment could cure a patient with a duodenal ulcer, then the ulcer should be present post mortem if no operation were performed, whatever the immediate cause of death might be. But Mr. Moynihan has found that duodenal ulcer is a comparatively common disease, whereas even the most recent statistics show that duodenal ulcers are found comparatively rarely post mortem, so that the conclusion is inevitable that the majority of cases recover completely without operation and eventually die from some totally different cause.

It is often asked how the thick mass around a chronic ulcer can possibly disappear under medical treatment, and Mr. Moynihan has told us that the conditions in the ulcer are, as a rule, such that nothing but surgical treatment could avail. But ulcers of this sort have been found to have completely disappeared when the abdomen has been opened several months after the performance of a gastro-enterostomy, and the mechanical and chemical effects of medical treatment are almost identical with what is supposed to occur after a gastro-enterostomy.

At one time I thought that a history of many years' duration was a definite indication for operative treatment, but a case recently under my care has made me change my opinion. A man, aged 50, had for twenty-two years suffered from typical attacks of severe hunger-pain, the last of which had continued with hardly an intermission for nearly a year. On several occasions hæmorrhage had occurred. The stomach was dilated and there was visible peristalsis. As the ulcer was of such long standing, and as it seemed to have given rise to definite obstruction, I advised operation. For business reasons the patient had to postpone the operation for four months, during which he was carefully dieted and took 1 oz. of olive oil before each meal. He quickly lost his pain, and the dilatation and visible peristalsis disappeared; but, with the long history, I still thought an operation advisable, and the patient was very anxious to have one done. Mr. Rowlands operated and found a definite white scar on the duodenum, but there was very little thickening, and he was able to convince himself that the ulcer had completely healed, although four months earlier it must have been active and the thickening round it sufficient to produce well-marked obstruction. Here then is a case which proves most definitely that even after twenty-two years a duodenal ulcer can heal as a result of medical treatment.

Everyone will admit that medical treatment during an attack is generally followed by complete relief, so long as no obstruction is

present. But the little-known conditions which led to the formation of the original ulcer may be unaffected, so that exposure to cold or a period of over-work may cause the healed ulcer to become active again or a new ulcer to form. On the other hand, gastro-enterostomy not only causes an ulcer to heal, but it renders a return of symptoms comparatively unlikely. This difference between the results of medical and surgical treatment is due to the fact that we are too much inclined to have done with a patient, and a patient is too much inclined to have done with us, immediately his pain has gone. But in truth our duty is only half done when the ulcer has healed, as we must still teach the patient how to avoid a recurrence.

In my opinion an operation should never be recommended, in the absence of obstruction, until thorough treatment by diet and rest in bed for at least four weeks has failed to give relief, or until frequent recurrences have occurred in spite of every precaution. This is not the place to enter into details of medical treatment, but I should like to say a few words on the prophylactic measures which should follow the recovery from an attack.

All mechanical and chemical irritation of the stomach and duodenum should be avoided. Both forms of irritation can result from bad teeth, and no case has been properly treated until the teeth have been put into perfect order and artificial teeth provided to replace any which are lost. I have hardly ever seen gastro-enterostomy performed for duodenal ulcer on a patient with sound teeth, and on one occasion the patient had only two carious stumps in his mouth; it is absurd to say that medical treatment had received a fair trial in such an individual. It is rare to find a patient with a duodenal ulcer, who has not habitually eaten too fast, and I believe that the bolting of food is the chief cause of the recurrence of attacks in times of mental stress. The food should be properly masticated, and the patient, when he is in a hurry, should eat a stick of chocolate or drink a glass of milk rather than swallow a half-chewed meal. The mechanical irritation produced by the completely indigestible parts of many fruits and vegetables should be avoided, and acid or highly seasoned foods and excess of alcohol and tea should be prohibited.

Whatever the relation may be between hydrochloric acid and duodenal ulceration, there is no doubt that the less free acid that reaches the duodenum the less likely is an ulcer to form. As carbohydrates produce an abundant secretion of hydrochloric acid, which, unlike proteins, they cannot neutralise, the quantity taken should be limited; $\frac{1}{2}$ oz. of olive oil—or, in the rare instances in which there is difficulty in

taking it, 1 oz. of cream—should be taken before each meal. For two and a half years I have been convinced of the efficacy of this method of diminishing the secretion of hydrochloric acid, and the recent chemical investigations of Dr. Craven Moore have put this treatment on a more scientific basis.

I can fully confirm Mr. Moynihan's observation that attacks are particularly frequent in cold weather, but, like Sir Lauder Brunton, I think that the autumn and early spring are even more dangerous seasons for patients with duodenal ulcer than winter, and damp weather is worse than dry and frosty weather. I have also observed that the majority of patients with gastric and duodenal ulcer suffer from cold extremities, and in many cases the onset of vasomotor disturbance immediately precedes the onset of digestive symptoms. Such patients often state that when the pain is worst their feet feel very cold, and putting the feet into hot water frequently relieves the pain. We have recently found that the onset of pain in duodenal ulcer is associated with a rapid and well-marked rise in blood-pressure, which is much greater than that observed in most other painful conditions. These facts suggest that vasomotor disturbances may be a factor in the production of ulceration, and it is therefore of great importance for patients, who have once had an ulcer, to take every possible precaution to prevent getting cold extremities.

With regard to the actual operation to be done in suitable cases, I cannot help thinking that the "ideal" operation of excision, as advocated by Mr. Moynihan, is irrational, not because a second ulcer may be present—for Mr. Moynihan would obviously do a gastro-enterostomy were this the case, but because this operation has no advantages over medical treatment, as it does nothing to diminish the likelihood of a recurrence by diverting the acid gastric contents from the duodenum.

I am very grateful to Mr. Moynihan for teaching us how frequent a condition duodenal ulcer is, and how easily it can be diagnosed. I am equally grateful to him for having put it in my power to convince him of the error of his views of the futility of medical treatment. A gentleman, who for eight years had had slight attacks of hunger-pain at long intervals, had an almost continuous attack during the eighteen months previous to last August. He wished to give medical treatment a trial before resorting to surgery, which was strongly urged upon him. Mr. Moynihan therefore very kindly sent him to me. He started treatment with oil and rest in bed on August 23; he was up again on September 17, and, in spite of the cold and changeable weather, he has had no

suspicion of hunger-pain since the day the treatment began. Clearly he is no proof of my contention yet; but I have every hope that by following the preventive treatment I have advised he will live to convince Mr. Moynihan that medicine can produce as complete and as lasting a cure as surgery.

Mr. HERBERT PATERSON said that he took it that the object of the discussion was to put on record the opinions of those who had had especial experience in the treatment of duodenal ulcer. This being so, he felt sure that Mr. Moynihan would understand that if he (Mr. Paterson) criticized some of the statements made in the paper which had been read, it was not because he did not thoroughly appreciate the great value of the work which Mr. Moynihan had done, but solely with the object of eliciting the truth. Mr. Moynihan had given them a very clear and concise account of the symptoms of duodenal ulcer, but in his (Mr. Paterson's) experience he had not found that the symptoms were anything like so uniform as had been described. There were, for instance, great differences in the way in which patients described their pain. In some cases the onset of pain was immediately after the ingestion of food; in others it was continuous, and in others it occurred only two or three hours after a meal. Dr. Hutchison had suggested that in some cases "hunger-pain" was the expression of a purely functional disorder, but, so far, in every case, in which hunger-pain was one of the symptoms, in which he (Mr. Paterson) had operated, some definite organic lesion, either of the duodenum, gall-bladder, or appendix, existed. There was one difficulty in diagnosis to which reference had not been made in the discussion—namely, the differential diagnosis between duodenal ulcer and gastric crises. He had seen two patients on whom gastro-jejunostomy had been performed for gastric crises, and naturally neither had been benefited by operation. He hoped that the physicians would give them some information on this point. In the one case which he had had an opportunity of investigating there was hyperchlorhydria during the gastric crisis, but absence of free hydrochloric acid after the attack had passed off. He had made accurate gastric analysis in twenty-eight of his cases of duodenal ulcer, and had found that in 60 per cent. there was a marked excess of free HCl. In some cases the amount was as high as 0.14, and in others as high as 0.04. In discussing this subject it was very necessary to define with exactness the terms they used. He was of opinion that the term hyperchlorhydria should be limited to those cases in which there was an excess of free

HCl, the term "hyperacidity" being used for those cases in which there was a high total acidity of the gastric contents. The divergence in the results of gastric analysis in cases of duodenal ulcer probably depended to some extent on the time at which the analysis was made. In some cases, at any rate, the hyperchlorhydria disappeared after an attack had passed over. In some cases it varied from day to day. In one case in which HCl was absent, a few days later 0.04 per cent. of free HCl was present.

Some of the speakers had talked of a functional hyperchlorhydria. He confessed that he had not sufficient "subtlety of mind" to appreciate the existence of a condition of purely functional hyperchlorhydria. In these matters one was naturally influenced by one's own experience. He had operated on nearly fifty cases of hyperchlorhydria, and in every one of them there was an organic lesion either of the stomach, duodenum, gall-bladder, or appendix. No one, so far as he was aware, had called attention to the fact that appendicular disease does, by causing secondary changes in the gastric mucosa, lead to marked hyperchlorhydria. He agreed with Mr. Moynihan that in cases of severe recurrent hyperchlorhydria there was no reasonable doubt as to the existence of a duodenal ulcer. Dr. Hutchison had referred to Harley and Goodbody's statistics as to hyperchlorhydria, but he would point out that the cause of the hyperchlorhydria was not investigated by operation, so that there was no evidence that the hyperchlorhydria was purely functional. It was quite possible that a duodenal ulcer in an early stage might be so small and accompanied by so little infiltration that it could not be recognized at operation from an external examination of the duodenum; but he wished most emphatically to dissent from the opinion expressed by one of the speakers that the duodenum or stomach should be opened and explored from within. Such a procedure was totally unnecessary and entailed unnecessary risk. If no ulcer could be found from an external examination of the duodenum, gastro-jejunostomy should not be performed and the abdomen should be closed. The risk of simply opening the abdomen was very slight, but if it went forth as having been said at that Society that it was justifiable to open and explore the stomach and duodenum from within, then the mortality from these operations would be materially increased. Dr. Hutchison appeared to regard melæna as a sign of duodenal ulcer, but he (Mr. Paterson) had observed several cases of severe melæna due to appendicular disease. Mr. Moynihan regarded hæmorrhage as evidence of deep penetration of a

duodenal ulcer, but he (Mr. Paterson) was of opinion that the hæmorrhage more often came from the mucous membrane of the stomach than from the duodenal ulcer itself. He had two sections of mucous membrane which definitely showed the oozing of blood from between the gastric cells.

Mr. Moynihan, in speaking of vomiting, said that it was generally due to stenosis. In his (Mr. Paterson's) experience it was more commonly due to spasm of the pylorus than to organic stenosis. These were the points on which he differed from Mr. Moynihan. When they came to the question of treatment, he was most thoroughly in agreement with him. The treatment of *chronic* duodenal ulcer should always be surgical, but to justify operation the attacks must be recurrent. He thought that this was a perfectly clear position to take up. There could be no doubt whatever that duodenal ulcers did heal under medical treatment. He had operated on three cases in which only a scar of an ulcer was found in the duodenum, but, notwithstanding this, the patient's symptoms had persisted until operation was performed. He thought that there was a certain amount of evidence that, although a *chronic* duodenal ulcer might heal, surgical intervention was still necessary in a proportion of the cases. It seemed that duodenal ulcer was a more intractable condition than gastric ulcer. He had shown elsewhere that, in the case of hospital patients at any rate, probably not more than 25 per cent. of patients with chronic gastric ulcer were permanently cured. He did not think, therefore, that they could expect the results of medical treatment of chronic duodenal ulcer to be very satisfactory. He did not wish to under-estimate the value of post-mortem evidence, but he maintained that an ounce of operation-table pathology was worth a pound of post-mortem pathology, and he rejoiced that many physicians now came and saw operations on their patients, and so could observe the pathological conditions present with the clinical features of the case fresh in their minds. Sometimes a duodenal ulcer was found unexpectedly at a post-mortem; the notes were looked up, but no history of duodenal ulcer was found recorded. This did not prove that the ulcer had existed without causing symptoms. They could not cross-examine a corpse. Even during life it was sometimes difficult to ascertain the exact extent of a patient's symptoms, as important symptoms were sometimes overlooked, forgotten, or the history of them purposely withheld.

He agreed that at the present time, when surgical treatment for duodenal ulcer was necessary, gastro-jejunostomy was the best available

operation. He had performed forty-one operations for duodenal ulcer—three perforated cases and thirty-eight non-perforated—with a mortality in the non-perforated cases of 2.6 per cent. Dr. Hutchison had laid stress on the importance of after-treatment. He (Mr. Paterson) thought that too little attention had been paid to this point, the importance of which he himself had repeatedly emphasized. He always advised his ulcer patients to keep on a milk diet for at least six months after operation, and longer if there was marked hyperacidity. As to the results of surgical treatment, in his experience from 85 to 90 per cent. of the patients were cured. They could not expect to cure all cases, even with the best surgery; and they must remember that some patients were not cured, not because the gastro-jejunostomy was a failure, but because, owing to their feeble state of health, they fell victims to inter-current diseases, such as phthisis. Further, some of the bad results after gastro-jejunostomy were due to its performance in the absence of definite organic indications. He wished to urge most emphatically that gastro-jejunostomy should not be performed on a clinical diagnosis, unless the operation findings showed the existence of organic disease which indicated operation.

It seems fated that the operation of gastro-jejunostomy should be associated with bogies. A few years ago we had the bogey that gastro-jejunostomy interfered with metabolism. That bogey was now dead, and he thought that he might claim to have performed the chief part at its obsequies. Now another bogey had come on the scene—namely, the notion that it was necessary to exclude the pylorus when performing gastro-jejunostomy in cases of patent pylorus, a procedure advocated by one of the speakers at the previous meeting. He believed this is to be a retrograde suggestion. The line of argument was that when the pylorus was patent, the food passed out through the pylorus, and the gastro-jejunostomy opening, being functionless, gradually closed up. He maintained that there was no ground for this belief. If gastro-jejunostomy were properly performed, and after-treatment efficiently carried out, there was no risk of the opening becoming closed. He had had a number of opportunities of observing after-results both in his own patients and in those of others, and he had never yet seen a case in which the opening had closed, except when the operation had been performed by obsolete methods. He was strongly of the opinion that exclusion of the pylorus was based on erroneous pathology, and should be considered meddlesome surgery.

Dr. NATHAN RAW said that the chief points to which he wished to draw attention were: First, the frequency of duodenal ulcer; and, secondly, the very difficult question of early diagnosis. Those of them who had the opportunity of admitting into hospital cases of perforated duodenal ulcer must all be grateful to Mr. Moynihan for having classified more clearly the very early symptoms of duodenal ulcer. He was impressed with the immense importance to the patient of an early and accurate diagnosis being made. They all knew that, if they had cases of serious duodenal ulcer, they would endeavour, if possible, to send them to Leeds and put them under Mr. Moynihan's care, thus proving that Mr. Moynihan might meet more cases of duodenal ulcer in his practice than he himself was likely to do in Liverpool. Out of over 40,000 admissions into his hospital during the last twelve years there had been 182 cases of gastric ulcer and 37 of duodenal ulcer. But, after hearing about the minute differences upon which diagnosis depended, he thought it possible that in some cases their diagnosis might have been wrong. Nevertheless, out of 5,000 autopsies, many of which he had himself performed, during the last twenty years, he had only come across thirteen ulcers in the duodenum. If the ulcers had healed as quickly as had been said, the post-mortem evidence could not go for much. But he felt sure from his experience that the number of cases of duodenal ulcer in Liverpool was not as great as appeared to be the case in Leeds. With regard to the class of people who were attacked with duodenal ulcer, it was known that gastric ulcer was most common among domestic servants; but his experience was that duodenal ulcer was very rare among domestic servants, and more common among males than among females. He asked whether Mr. Moynihan could give any explanation of the latter fact. A fortnight ago he had a case in the hospital of a man who presented all the classical symptoms which Mr. Moynihan described in his last paper, and he said here at least was a case of duodenal ulcer: there were marked hunger-pain, great pain after food, &c. But, though a careful post-mortem search was made, there was no ulcer, but a chronic thickening of the pylorus, which would explain all the gastric symptoms. So he agreed that the symptoms which Mr. Moynihan described might be present in other conditions besides the classical ones of duodenal ulcer.

Dr. CRAVEN MOORE expressed his keen appreciation of the work of Mr. Moynihan on the subject under discussion, and, although he did not see eye to eye with him, he was fully conscious of the debt which the

profession owed to that gentleman for the demonstration of the frequency with which ulcer of the duodenum occurred in association with the symptom-complex described. He could not agree that those symptoms were, as stated by Mr. Moynihan, unassociated with a very definite disturbance in the functional activity of the stomach, to which, however, the term "hyperchlorhydria"—a conception based on an inadequate knowledge of gastric physiology—appeared to be inapplicable. His own experience—based on the investigation of cases in which the anatomical condition had been examined at operation—definitely established the occurrence of a hypersecretion of gastric juice in association with ulcer of the duodenum. Of seven recent cases, it was found that hyperacidity of the stomach contents after a test breakfast (plain tea 10 oz., four breakfast biscuits) obtained in six, and in the remaining one the values were about the normal superior limits; the average acid values in seven cases were: free HCl 50, total acidity 70. That such hyperacidity of the stomach contents was a factor in the causation of the symptoms would appear from the relief which was afforded by dilution, or neutralization; that it was not the sole factor was well recognized. As explaining the peculiar time-relation of the so-called hunger-pain, it had not been sufficiently realized that when hypersecretion obtained, as it did in duodenal ulcer, and also apart from that lesion, the acidity of the stomach contents increased as the stomach emptied itself, the final portions to leave the stomach approximating to undiluted gastric juice.

Again, from his experience he could not believe that the symptom-complex described was so exclusively significant of ulcer of the duodenum as had been maintained by Mr. Moynihan. In a series of cases in which these manifestations had been so typically present that a diagnosis of duodenal ulcer had been made, laparotomy had revealed a normal duodenum, with (1) ulcer of the pylorus, (2) chronic calculous cholecystitis, and (3) atonic dilatation of the stomach—*myasthenia gastrica*. In one of the latter the patient said spontaneously that as a child she used to speak of her discomfort as "hungry pain." Accordingly he had come to regard the symptom-complex described as signifying the existence of a disordered secretory activity of the stomach, associated probably with pyloric spasm, and to such the term "acid dyspepsia" might well be applied without prejudicing the presence or absence of an associated anatomical lesion. In order that the existence of an ulcer of the duodenum in such a condition might be definitely established, it was necessary to determine the presence of a localized area of tenderness

above and to the right of the umbilicus, it might be with some degree of muscular rigidity, but without hyperaesthesia, and the occurrence of occult hæmorrhage, during an attack. Occult hæmorrhage is a sign of considerable value; in duodenal ulcer it occurs intermittently, and in his own cases was demonstrated in 100 per cent.

Mr. C. P. CHILDE (Portsmouth) said he thought the practical and important point which they had to decide was when cases of duodenal ulcer should be handed over to the surgeon. It was difficult to determine, because patients without organic disease might suffer from symptoms which were indistinguishable from those of duodenal ulcer. Mr. Eve, Dr. Raw, and Dr. Moore had each mentioned such cases. As a practical surgeon, he could say that a large number of cases of duodenal ulcer went on for years, the patients being chronic invalids before they were diagnosed to have the condition at all. That was a state of things which it was sought to eradicate, and which Mr. Moynihan had done so much to help them to do. Mr. Moynihan had operated on 200 cases of duodenal ulcer, and in eleven cases in which it had perforated. That showed that, at all events, he had been able to get the cases earlier than most surgeons. He (Mr. Childe) had operated upon about thirty cases, and in no fewer than seven of them there was perforation. He would mention three cases which occurred in his practice last year, all cases of perforation. They were well-to-do people, and had had abundance of medical advice. All had suffered during many years from symptoms of duodenal ulcer, yet none had had it suggested to them that that was their condition. After perforation of course they came to operation, and ulcers were found probably of an age which corresponded with their symptoms—in one case seventeen years, in one fifteen, in the other many years. Mr. Moynihan had done well to relegate hæmorrhage to a subordinate position in the symptoms—not the minute hæmorrhage alluded to by Dr. Moore, but hæmorrhage which the patient noticed. In not one of the seven cases of perforation on which he had operated had such hæmorrhage been noticed. One of the three cases he had mentioned was that of a medical man who had lived in Portsmouth seventeen years and had long been a chronic invalid from indigestion, but was not supposed to be suffering from a duodenal ulcer. Recently he began to have symptoms of dilatation, vomiting large quantities of food at long intervals. On the advice of the practitioner who was attending him, he consulted a physician to a large London hospital, who said he was suffering from atonic dilatation of the stomach and had

no duodenal ulcer. Within three months he perforated, and there could be no doubt that he had had duodenal ulcer for many years. His experience had been largely with the disease at a late period, whereas Mr. Moynihan seemed to have been more fortunate. As things were at present, many patients seemed to go on without any serious attention being paid to their complaint until they came to some catastrophe, such as hæmorrhage or perforation. The oldest patient on whom he had operated was aged 66, and for thirty-five years he had been suffering from symptoms of duodenal ulcer. He then for the first time had severe hæmorrhage. A tumour was easily felt, and he was operated upon by closure of the perforation and gastro-anastomosis four years ago, and had remained well ever since. The result showed that the complaint was not malignant, and he probably had had a duodenal ulcer for very many years. In the late cases he did not think there was any operation which was feasible except gastro-enterostomy. But he agreed that in the early cases, if one could be sure the ulcer was single, a better operation was excision. If anastomosis were done in an early case, while the ulcer was active, the gastric contents would pass by the anastomotic opening and the ulcer would probably then heal; afterwards, although the anastomotic opening might not close, he did not see why the food should not pass again by the pylorus and the condition recur. So, if the ulcer were small and single and there were no obstruction at the pylorus, he would excise the ulcer. If gastro-jejunosomy were performed in the early stage, he thought that extended experience would very likely show that it would be necessary to close the pylorus with a view of preventing a recurrence of the trouble.

MR. MOYNIHAN, in concluding the debate, said that it was now a matter of general agreement that the symptoms which he had described were dependent for their presence upon a structural lesion—ulcer of the duodenum. They were at last escaping from the bondage of the vocabulary and were ceasing to apply incorrect and misleading terms, such as "acid dyspepsia" and the like to an organic disease. The practitioners who were still submissive to the tyranny of the ancient text-books often quoted these authorities as being opposed to his opinions. It was true that these works contained no recognition of the facts that duodenal ulcer was a common disease, marked out by the plainest symptoms and admitting of easy diagnosis. Recent authors, however, were adapting their views to the wider knowledge with which

the surgeon had lately equipped them. Precise inquiry into all the details of the anamnesis prevented a majority of the mistakes in diagnosis to which reference had been made. One of the difficulties confronting an author to-day was the desire of many readers for the most concentrated epitome of his message. The torrent of Niagara, they seemed to say, must be described in terms of the pipette. So "hunger-pain," a term which had been debased and shorn of all the attributes with which he had clothed it, was held to indicate undoubtedly the presence of a duodenal ulcer. All the varied qualities of this important symptom, however, required consideration before a diagnosis based upon it could have any substantial value. We were entitled to say that by a careful study of the anamnesis, and from that alone, the diagnosis of duodenal ulcer could now be made with a degree of accuracy not exceeded in the case of any other abdominal disease. The physical signs were few and of no real value in enabling a diagnosis to be made. Tenderness, muscular rigidity, and increased epigastric reflex on the right side might one or all be present, and, if present, were better marked during the time that pain was present.

The only difficulties in diagnosis were concerned with the differentiation of duodenal ulcer from gastric ulcer, from gall-stones, and from appendicitis. If an ulcer were not found on palpation and inspection of the stomach or duodenum, inspection of the mucosa was useless. He held very confidently to the opinion that an ulcer which had been the cause of protracted and recurring symptoms had always reached the outer coats of the stomach or duodenum; it was never restricted to the mucosa. Instead of the unnecessary search within the stomach, the gall-bladder and appendix should be examined. The condition of "appendix dyspepsia" was important and as yet not generally recognized.

If an operation were undertaken for the relief of long-standing symptoms believed to be due to a duodenal or gastric ulcer, and no ulcer were discovered, it was absolutely necessary that the appendix should be examined; in a notable proportion of such cases it would be found adherent and obstructed, and its removal would be followed by a disappearance of all symptoms. It was quite inadmissible in such cases to perform gastro-enterostomy. The great fault had been that gastro-enterostomy had been done because the diagnosis of duodenal ulcer was made before the abdomen was opened. But the operation should not be done unless a definite reason for it existed. Some surgeons seemed to think it was a humiliation for them to open an

abdomen after making a diagnosis and not to discover the particular condition which they had supposed to be present. He (Mr. Moynihan) had no compunction in setting his diagnosis aside as soon as it was found wrong, and going in search of something else. There was the perennial example of Saul, who went in search of his father's asses and found a kingdom. He emphasized the point that gastro-enterostomy should never be performed unless there was a demonstrable lesion which called for it. When fault was being found with the treatment of duodenal ulcer which had never been present by gastro-enterostomy, one must not attribute to the operation faults which were those of the operator.

The different views in respect of treatment held by physicians and surgeons would probably disappear as it became more widely recognized that the symptoms pointed to an organic and not to a functional disorder. If medical treatment in early attacks proved powerless to prevent recurrence, then surgical treatment would admittedly be necessary. If the ulcer were small and single, excision would cure the patient; if the ulcers were large or multiple, as was generally the case, gastro-enterostomy would be necessary. Many of the early unsatisfactory results of gastro-enterostomy were due to the wrongful application of this operation, the precise indications for which were now fully understood. After the operation the patients required supervision for some time.

Surgical Section.

February 8, 1910.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

The Radical Operation for Cancer of the Pylorus, with especial reference to the Advantages of the Two-stage Operation and to the Question of the Removal of the Associated Lymphatics.

By ERNEST W. HEY GROVES, M.S.

THE very remarkable advance which has been made in the radical treatment of cancer of the stomach during the thirty years since Billroth first successfully performed it is one of the most notable achievements of modern surgery. I remember very well, as a student at St. Bartholomew's Hospital fifteen years ago, hearing Mr. Butlin say that pylorotomy for cancer was an operation of such great dangers and poor results that it was scarcely a justifiable procedure; and in those days the surgeon who attempted even a gastro-enterostomy was regarded as one who ventured upon dark and mysterious paths. The stomach and intestine, united by various mechanical devices, were carefully preserved in the Museum after the autopsy as a warning to the rashly adventurous! And yet, now, successful gastro-enterostomy by simple suturing is an every-day occurrence, and the recoveries from gastrectomy for malignant diseases can be numbered by the hundred.

But, nevertheless, all who read and work at the subject of cancer of the stomach must be much more impressed with the need there still is for improvement in the treatment than with the improvements which have already been effected. So few patients with this disease are ever

offered the chances of radical treatment, and of these few the majority are too advanced in disease to have more than a palliative operation performed. Of those who actually have their disease removed, a considerable proportion die as the result of the operation, and, of those who recover, many succumb to recurrence almost as soon as if they had never been treated.

If we concentrate our attention upon the few ideal cases which come to the surgeon in good time, we are impressed with the splendid thing that surgery can do for cancer of the stomach. But if we take a large view of the disease in the aggregate of all its victims, we must be depressed by the very little that is done. The reasons for this deplorable state of affairs is not far to seek. The blame—if blame there be—attaches almost equally to the public, the general medical practitioners, and to the surgeons. The public and the medical practitioners do not believe in the possibility of radical cure of the disease. Consulting physicians waste valuable time in employing various recondite methods of diagnosis. The surgeon is too often only called in as a last desperate resort, when neither sophistry nor science can any longer doubt that the patient is in the clutch of a rapidly fatal disease. And the surgeon, with the few cases that do eventually reach him, is tempted to perform palliative operations on patients that had better be left to the solace of morphia, and to attempt radical operations in patients too ill to recover from them, or in whom total removal of the diseased area is impossible. And the physicians, general practitioners, and public are likely to be more impressed by the deaths and recurrences than by the successes, especially as the former tend to confirm their adverse prejudices against surgery.

My object in narrating all the cases in which I have performed the radical operation for cancer of the stomach, and in collecting the results of other writers, is to emphasize the following points:—

- (1) That there is a good prospect of cure in early cases;
- (2) That exploratory operations should be performed for diagnostic purposes in all doubtful cases;
- (3) That the immediate operative mortality may be greatly reduced by operating in two stages;
- (4) That a more systematic attempt should be made to remove the whole of the associated lymphatic area;
- (5) That the limits of operability, both for palliative and radical treatment, should be more rigidly defined, so as to lessen, if possible, the number of surgical failures.

It will be convenient to give a short account of my own cases before commenting upon them or upon the general subject.

CASE I.

H. B., coal carrier, aged 57, admitted to the Bristol General Hospital July 27, 1903, for pain and vomiting. Family history: Father died when aged 56 of a "stone cancer in the neck." History: Eight months ago began to have pain after meals, followed by vomiting and loss of flesh. Condition: Considerably emaciated; nothing seen or felt abnormal in the abdomen. Stomach washed out and about $1\frac{1}{2}$ pints of balmy fluid withdrawn. Free hydrochloric acid absent. Vomiting occurs about eight to twelve hours after food. No hæmatemesis.

Gastrectomy, July 29, 1903: A hard white mass involved the pylorus and extended about 1 in. from it. The involved area, with about 1 in. of healthy stomach and intestine, was removed and the stomach joined to the duodenum over a bone bobbin. At the end of the operation the patient was almost moribund, and for nearly twelve hours transfusion, enemata, brandy and strychnine were scarcely able to keep him alive.

Subsequent course: For three days his progress was fairly good. He was fed chiefly by the rectum, but began taking well by the mouth. On August 2, in the afternoon, he suddenly developed symptoms of peritonitis, the pulse running up from 108 to 132, and he died on August 3. No autopsy was allowed.

Microscopically, the growth was an adeno-carcinoma. The cause of death was probably leakage at the line of suture.

CASE II.

A. H., woman, aged 56, sent to the General Hospital by Dr. Taylor in September, 1906, complaining of pain after food and vomiting. History: Married woman with seven children, the youngest being aged 17. She had good health until about a year ago, when she began to suffer with "indigestion." About a fortnight ago she vomited, and this has continued every day since in large quantities, there being sometimes a little blood. She has rapidly lost flesh lately. Condition on admission: She is a thin, dry-skinned woman, nervous, bright, and intelligent. All the organs are normal except the abdomen, where a hard mass can be felt just above and to the left of the navel. It moves with respiration. There is no evidence of gastric dilatation.

120 Groves: *Radical Operation for Cancer of Pylorus*

First operation, September 7, 1906: Median incision. A hard nodular mass found on the lesser curvature of the stomach near the pylorus. No adhesions or enlarged glands. A posterior gastro-jejuno-stomy by direct suture with two rows of continuous thread stitches. She had a little vomiting after the operation, but since then has been taking fluids well and making good progress.

Second operation, September 14, 1906: Partial gastrectomy. Both stomach and duodenum were divided between two clamps after ligation of the vessels in the curvatures, and then each was closed by a double row of sutures. Shock was slight. Part removed consists of the pyloric end of the stomach, including 4 in. of the lesser curve and 5 in. of the greater curve. A nodular growth is projecting into the lumen of the pyloric canal and blocks it completely. It begins abruptly at the duodenal end of the stomach and extends 2 in. along the lesser curve and 1 in. along the greater. Portions of the less and great omenta are attached. Microscopically, the growth was a spheroidal-celled carcinoma.

After-progress was perfectly satisfactory, and she left the hospital within one month. For about seven months her health was good and she gained 16 lb. in weight, and could eat ordinary food without pain or vomiting.

June 1, 1907: Saw her with Dr. Taylor on account of an obscure illness. She lay in bed in a very drowsy condition, saying she felt very bad. There were absolutely no physical signs of growth or other disease in the chest or abdomen. Urine was scant and albuminous. Some soft swellings were present on both sides of the neck, but these were very indefinite and did not feel like glands. For six weeks longer she suffered from symptoms of cystitis, retention of urine, and uræmia, from which she died on July 10, 1907. No post-mortem was allowed.

CASE III.

F. W., aged 66, sent to the General Hospital by Dr. Blackley on September 2, 1906, complaining of pain after food. History: Loss of flesh (amounting to about 1 st.) and appetite since the beginning of the year. Pain of a moderate character for last three months. Vomits a large amount occasionally. No bleeding. Condition: Healthy-looking and well-nourished man, of good physique. Well-marked dilatation of the stomach, which easily holds 4 pints of fluid. When distended with air, the greater curvature reaches 3 in. below the navel. A well-marked peristaltic wave from left to right across the gastric area. Fig-seeds

retained in the stomach for twenty-four hours. Free hydrochloric acid absent. An indefinite resistance is felt to the right of the mid-line above the navel.

First operation, September 12: A growth was found almost confined to the pylorus. A very remarkable tortuous and dilated condition of the mesenteric arteries and branches of the celiac plexus existed. A posterior gastro-enterostomy by double-thread suture. The opening was made through the meso-colon to the right of the middle colic artery. He made a good recovery from this except that on the second day there was a rather severe hæmatemesis, which was stopped by $\text{m} \times$ of adrenalin chloride (1 in 1,000), given in water by the mouth.

Second operation, September 19: Partial gastrectomy. The only noteworthy point was that the anastomosis opening had been made too near the pylorus, so that the gastric incision could not be made as far from the growth as was desirable. He made a rapid and complete recovery.

Mass removed consists of the much-thickened pylorus with about 1 in. of healthy gastric and duodenal mucous membrane at each end. The circular muscle is much hypertrophied, as seen in longitudinal section through the part removed. The pyloric outlet is practically completely occluded. The growth itself is seen in section as a white sclerosing scirrhous sharply ending towards the duodenum, but fading off towards the stomach gradually.

After history: Between November, 1906, and November, 1907, his progress was uninterrupted; he increased from 10 st. to 11 st. 12 lb. On November 25, 1907, he suddenly developed jaundice without any pain, and this gradually became more serious. In the beginning of 1908 a weeping sore formed at the umbilicus, and soon after a nodular growth could be felt beneath the scar. Considerable bleeding took place from this spot and melæna became well marked. Death occurred on April 15, 1908—nineteen months after the operation.

CASE IV.

J. M., aged 55, admitted to the Cossham Hospital on August 27, 1907, under the care of Dr. Nixon, suffering from abdominal pain and vomiting. History: Has had pain after food for the last five years. Vomiting began one year ago, and has been much more frequent during the last few months, during which time he has lost flesh rapidly. Condition: Looks extremely ill and emaciated. The stomach cannot

122 Groves: *Radical Operation for Cancer of Pylorus*

be definitely outlined, but at times there is a peristaltic wave from left to right. Just above the umbilicus to the right there is an area of resistance. He vomits about once each day, bringing up a large quantity at a time. Fig-seeds and grape-stones taken on August 31 were recovered from the stomach washing on September 2. Free hydrochloric acid absent.

First operation, September 6, 1907: The stomach was greatly dilated and dropped, so that the lesser curvature lay only just above the umbilicus. A hard nodular mass occupied the pylorus. A posterior gastro-enterostomy was done, and a piece of the anterior part of the pyloric mass removed in its whole thickness for microscopical examina-

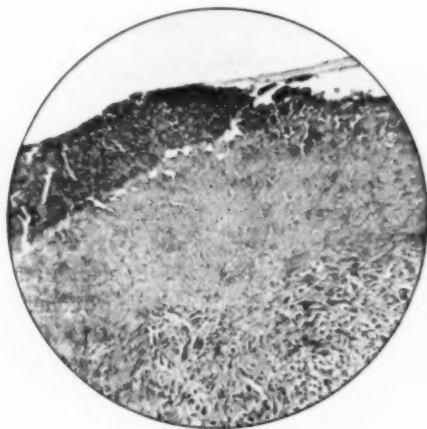


FIG. 1.

Section of the inner surface of the anterior part of the pylorus from Case IV.
The chronic inflammatory changes of a gastric ulcer are well shown.

tion. He recovered rapidly from this operation. Sections of the piece removed (fig. 1) showed nothing but fibrous tissue in its main mass and a piece of mucous membrane, which evidently formed the edge of a chronic ulcer. In spite of this, however, I was very doubtful as to the possible early malignancy of the growth as it had felt so hard and nodular. I therefore fully explained the matter to the patient, who unhesitatingly decided to have it excised.

Second operation, September 18, 1907: The pylorus, together with about 4 in. of the adjacent stomach-wall and 1 in. of duodenum,

was excised, the remaining parts of the stomach and duodenum being entirely closed. From the posterior part of the pyloric mass sections were taken, and these showed an early malignant infiltration of the stomach-wall, in the region of the dense scar tissue of the former ulceration (fig. 2).

Recovery was uninterrupted as far as the wound was concerned, but he often had attacks of pain and vomiting, bringing up from 1 pint to 2 pints of fluid. He made decided progress in spite of this, gaining several pounds in weight each week. I thought that the vomiting must be due to the downward sagging of the stomach, which now had no pyloric attachment. Probably this displacement temporarily kinked the

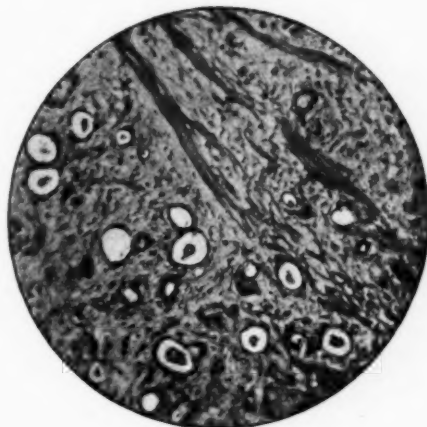


FIG. 2.

Section of the posterior part of the pylorus from Case IV. The muscular wall of the stomach is invaded by irregular epithelial acini. A very early stage of carcinoma.

anastomosis opening. However this may have been, the symptoms gradually disappeared, and he since has been in good health. In answer to a letter of inquiry written on June 8, 1909, he says that he is entirely free from pain, and that he has no vomiting. He can take ordinary food, but that it must be soft and well cooked. If he takes hard, lumpy food, it is apt to cause pain and vomiting. He is able to do light work as a gardener, and concludes: "I and my friends reckon it almost like a miracle; I am like one brought back from the dead and restored to life

and health again." His weight, which was 5 st. 7 lb. on leaving the Hospital, is now 8 st. 5 lb.

CASE V.

J. V., aged 32, a labourer, was sent to me by Dr. Llewellyn at the Cossham Hospital on August 12, 1908, suffering with pain and vomiting. History: No previous illness. For some months he had had pain after food, and for six weeks vomiting in large quantities has occurred every evening. Condition: A thin, well-built man with rather a yellowish tinge in his complexion, which he says he has always had. A small hard mass is occasionally felt under the right hypochondrium, but it often disappears. On August 17 a distinct contraction wave was seen and felt in the epigastrium. He vomits between 2 pints and 4 pints of fluid at a time, nearly every day, and it is noticed that the lump is only felt just after the stomach has emptied itself. When the stomach is filled with air it extends midway between the navel and pubes. Free hydrochloric acid is present in the gastric contents.

First operation, August 21, 1908: The stomach was much dilated, and the pylorus occupied by a hard growth evidently of malignant nature. A posterior gastro-enterostomy was performed by simple suture. He recovered well from this, and was able to take fluid food without sickness.

Second operation, September 1, 1908: The growth was excised with the neighbouring parts of the duodenum and stomach. The after-course was uneventful, and he made rapid progress. He weighed 9 st. 7½ lb. on his discharge on September 24. He is now at work as a cowman, and he can eat anything, having neither pain nor vomiting. He weighs 11 st. 3 lb. (June, 1909), and has grievously disappointed his speculative friends who had taken out a policy on his life!

Microscopical report: The deep layer of mucous membrane is transformed into typical adenoid cancer, and tubules of the growth penetrate deeply into the muscular tissues. There are also a number of large spheroidal epithelial cells, especially in the submucous tissue, like those of spheroidal-celled carcinoma.

CASE VI.

E. B., aged 38, married woman, sent to me at the Cossham Hospital on September 2, 1908, by Dr. Llewellyn, suffering from pain and vomiting. History: She has had six children, the last four-and-a-half

months ago, since which time her present symptoms began. Constipation was first noticed, and it has become more obstinate lately, so that she goes for several weeks without an action of the bowels. Vomiting set in soon after the constipation, and has also increased, so that lately she vomits everything she has taken. Pain in the epigastrium occasionally. She has lost $1\frac{1}{2}$ st. in weight during the past two months. Condition: Thin and very miserable woman. Her complexion is a yellowish-brown. The abdomen is slightly distended. A movable mass, about the size of a hen's egg, felt on the right linea semilunaris on a level with the navel. This is evidently in the pylorus, as its position changes when the stomach is distended. Free hydrochloric acid was present. The patient's disposition was gloomy and despondent in the extreme, and at first she refused any operation, but then consented, saying she was sure she would die. Feeling sure that she would never submit to a second operation, I decided to make the anastomosis and excision at the same time.

Operation, September 11, 1908: Posterior gastro-enterostomy by simple suture, followed by removal of the pylorus, which was the seat of a malignant growth, together with a large part of the stomach and a small piece of the duodenum. The whole procedure occupied one hour and a half, and she had much shock at its close; but she rallied from this, and continued to do well for six days.

On September 17 she began to vomit slightly, but this continued and became worse, and for the next four days she was constantly sick, bringing up a few ounces at a time of bile and mucus. Stopping food by the mouth, and feeding by the rectum, had no effect upon the sickness. I thought there must be some kinking of the intestinal loop concerned in the anastomosis, and I would have operated again, but that the patient refused and said she wished to die. On September 20 she consented, and the abdomen was re-opened. There was no peritonitis, and all appeared normal. The efferent loop of the jejunum was rather sharply bent at the junction of the stomach, and I therefore made a communication between the afferent and efferent limbs of this loop. After this the vomiting ceased, but she became more melancholic than ever, and there was the utmost difficulty in getting her to take any food at all. Her mind became quite deranged, and she was occasionally restless. She gradually sank and died on September 28.

Microscopical report: Section shows great thickening in the sub-mucous tissue composed of small round cells thickly sewn with large round epithelial cells. The latter penetrate the hypertrophied muscle

layer throughout its entire thickness. The surface of the mucous membrane over the growth in the submucous tissue is intact and normal. Spheroidal-celled carcinoma.

CASE VII.

R. H., aged 54, always in good health until twelve months ago, when he began to suffer from "indigestion." During the last six months there has been vomiting after all kinds of food, at varying intervals of six to twelve hours after meals. Pain has been dull in the epigastrium, and a little in the left shoulder. Lost weight to the extent of 28 lb. in six months—3 lb. in the last week. No hæmatemesis or melæna.

Present condition, June 7, 1909: Patient is very emaciated, weighing 7 st. 9 lb. In the epigastrium there is a well-marked tumour, rather smaller than a fist, and varying in consistency from time to time. It is distinctly hard and nodular at its right extremity, the left being smooth and elastic. The nodular part was regarded as the pyloric growth, and the smoother portion the hypertrophied antrum pylori. This proved to be correct at the operation. The whole mass moves with respiration, descending below the umbilicus. The stomach contents showed an absence of free hydrochloric acid, presence of lactic acid and of numerous Oppler-Boas bacilli. On washing out the stomach, which held 3 pints, the fluid was ejected with great force, due, no doubt, to the muscular hypertrophy of the organ.

June 12, gastro-enterostomy: Median incision. There was considerable shock on handling the viscera, the pulse becoming imperceptible. This improved after strychnine. A hard nodular mass was found occupying the pylorus and infiltrating the whole lesser curve of the stomach. Several hard glands were felt in the small and great omenta. No adhesions or growths in the liver. A posterior gastro-enterostomy was performed, the loop of jejunum being brought through the transverse meso-colon and through the gastro-colic omentum to the stomach. Double-continuous-thread sutures. A quart of hot saline was poured into the peritoneal cavity before closing the abdomen with through-and-through silkworm gut sutures. After-progress was all that could be desired. The patient was soon able to take soft food and had no vomiting.

June 24, partial gastrectomy: Incision re-opened. The great omentum was adherent by its tip to the root of the mesentery near the cæcum. This was broken down. The small omentum was divided just

below the liver and the coronary artery divided between clamps near its origin just above the pancreas. It was difficult to secure, owing to the large glands in its neighbourhood. The great omentum was then turned up and separated from the front of the transverse colon up to the gastro-enterostomy opening. The peritoneal base of the great omentum was next stripped off the upper surface of the transverse meso-colon and from the front of the pancreas. The stomach and duodenum were cut through between forceps, and the whole mass removed after ligature of the gastro-duodenal and pyloric vessels. The duodenum was closed by a double purse-string suture. It was necessary to remove a further part of the stomach, owing to the presence of infected glands in the lesser curvature. The section of the stomach extended from the right of the œsophagus to the middle of the great curve. The stomach opening was closed by double-continuous-thread sutures. Many bleeding points had to be tied over the head and anterior surface of the pancreas. After saline transfusion into the peritoneal cavity the pulse revived, and the wound was closed with gauze dressing. Patient died about two hours after the operation.

The parts removed are shown in figs. 3 and 4. The specimen consists of about 1 in. of duodenum and 4 in. of stomach. The wall of the latter is densely infiltrated with new growth, which is most prominent along the lesser curve, where it forms a nodular mass beneath the mucous membrane and on the peritoneal surface. A further portion of stomach was removed towards the œsophagus in order to get beyond the new growth in this position. Several involved glands were present in the lesser curve. The posterior view shows the peritoneum stripped off the head of the pancreas, which is connected to the root of the great omentum. Microscopically, the growth is a spheroidal-celled carcinoma with a large proportion of fibrous tissue. The coronary lymph-gland, the tip of the great omentum, and the root of the mesentery contained similar growths (figs. 5 and 6).

CASE VIII.

G. W., aged 49, haulier, admitted to the General Hospital under Dr. Symes on September 6, 1909, complaining of pain in the abdomen, vomiting, and loss of weight. He had no previous illness or dyspepsia. Symptoms began about three months ago with an attack of vomiting, since which time he vomited constantly, generally at night. The pain is chiefly on the right side of the abdomen, and is irregular in its

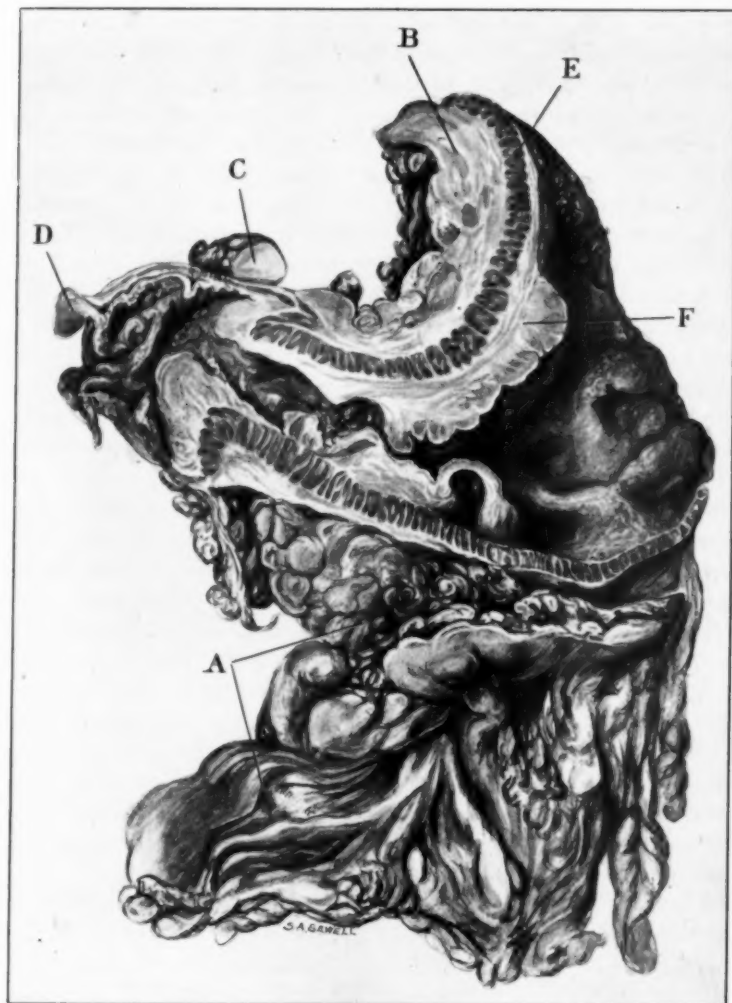


FIG. 3.

A part of the specimen removed from Case VII. A, great omentum; B, subserous growth; C, infected gland over the pylorus; D, duodenum; E, hypertrophied muscle layer; F, submucous growth. A further piece of the stomach was removed near B in order to get beyond the margin of the growth. (Two-thirds natural size.)

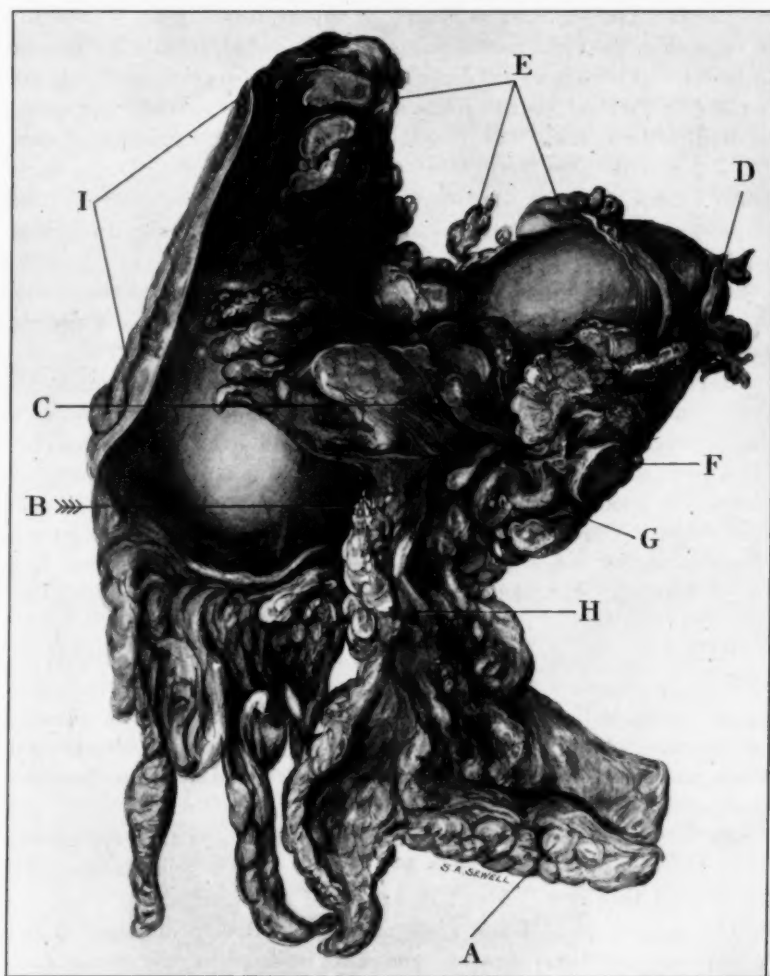


FIG. 4.

The parts removed from Case VII seen from behind. A, great omentum (cut short); B, points into the cavity of the lesser sac of peritoneum; C, peritoneum from the front of the pancreas; D, duodenum; E, infected glands at the lesser curve; F, one of the sub-pyloric glands; G, gastro-duodenal artery; H, peritoneum joining base of great omentum to the layer in front of the pancreas; I, cut distal end of the stomach. (Two-thirds natural size.) This is an actual representation of the parts removed by the method described at the end of the paper. The tissues at C, H, and A were all full of cancer, but would have been left behind by the usual operation.

occurrence. The abdomen is normal in appearance, slightly tender over the right side, the right rectus being held somewhat rigid. No tumour can be felt. There is a good deal of stomach splashing. Gastric contents after a test meal show the following: Total acidity, 0.438 per cent.; free hydrochloric acid, 0.0146 per cent.; combined hydrochloric acid, 0.0292 per cent. No lactic-acid or Boas-Oppler bacilli. Diagnosis of gastric cancer made on account of the rapidity of development of the symptoms, the comparative slowness of the pain, and the diminished hydrochloric acid.

September 22, operation: Posterior gastro-enterostomy, with removal of a hard mass from the under surface of the liver. A nodular growth occupied the back of the pylorus and extended about 1 in. along the lesser curvature of the stomach. The pylorus was adherent to the liver. On separating these adhesions a hard, nodular mass was found lying apparently in the longitudinal fissure of the liver. It was removed with a piece of the liver tissue, and a posterior gastro-enterostomy performed. A good deal of oozing from the hepatic wound necessitated a gauze drain. The mass removed consisted of dense fibrous tissue and calcareous stones. A subsequent leakage of bile proved it to have been the gall-bladder. He made a rapid recovery, and by September 30 the wound had healed.

October 8, opening and draining an abscess: On opening the abdomen to complete the operation a small collection of non-offensive pus was found at the site of the gall-bladder resection. This was packed and further procedures abandoned. From the pus a *Streptococcus faecalis* was grown in pure culture, this having probably come from the stump of the gall-bladder.

October 22 (one month after gastro-enterostomy), partial gastrectomy. There was considerable trouble in separating adhesions between the pylorus and the liver. About one-third of the stomach was removed with the greater part of the great omentum. Several enlarged glands were present in the omentum and also in front of the head of the pancreas. These were all removed. The stomach and duodenum were separately closed. Wound closed, a cigarette-drain being left in the upper angle. Recovery was good. There was some leaking, after the drain was removed, of fluid which excoriated the skin (? leaking from the pancreas or duodenum). The patient was able to eat well without any pain. The wound was healed by November 7, and he left hospital on November 12. His weight on December 7 was 9 st. 5 lb., as compared with 7 st. 11 lb. before operation.

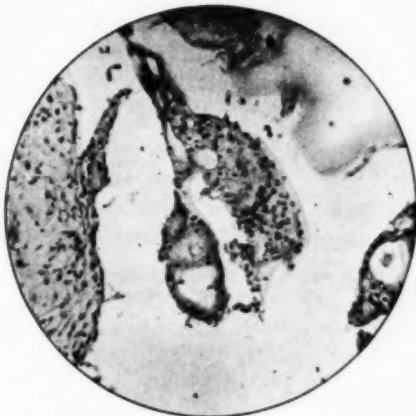


FIG. 5.

Section from the extremity of the omentum (Case VII) showing a mass of adenoid cancer in a large lymph-space.

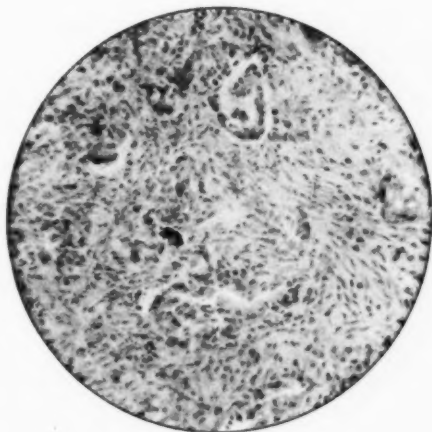


FIG. 6.

Section of the portion of mesentery (Case VII) to which the omentum was adherent, showing the invasion by malignant acini.

The specimen removed consists of the pylorus and part of the duodenum, being $4\frac{1}{2}$ in. in length. The great omentum is attached to the lower border of the specimen. There are several enlarged glands in the greater and lesser curves of the stomach. The pylorus is thickened and nodular, and its orifice is practically closed. The pyloric thickening runs along the lesser curve for about 1 in.

Microscopically all the coats of the stomach are thickened with much round-celled infiltration. No evidence of malignancy could be found in the section taken nor in the glands. Possibly, therefore, this was merely a simple ulcer, or possibly we have not yet discovered microscopically the malignant area.

ÆTIOLOGY.

Age and sex are factors of uncertain import in cancer of the stomach. The great majority of the patients are men aged between 40 and 60, but female and young cases are frequent.

My series include six men and two women, and all were well over 50 except two cases—one man and one woman. Makkas [35] gives the ages in 167 cases submitted to resection as:—

Between 20 and 30 years	6 cases
" 30 " 40	"	35 "
" 40 " 50	"	66 "
" 50 " 60	"	49 "
" 60 " 70	"	11 "
				<hr/>
				167 "

The exact relation between chronic ulcer of the stomach and cancer is difficult to determine. If the clinical history is taken as the criterion of the pre-existence of an ulcer then about one-eighth of the cases present it. Stumpf [46] described its occurrence in six out of forty-seven cases which were submitted to operation (12 per cent.). In my fourth case, a typical instance of this condition occurred in a man aged 55, who had for five years suffered all the tortures of ulcerative dyspepsia. At the time of the operation the malignant process was so limited that only at one spot out of three examined was it found. But, apart from these undoubted cases of ulcers which long run a chronic course and finally become malignant, there are two other conditions in which simple ulceration and cancer are associated or confused. On the one hand, an ulcer may become malignant very soon after its origin. This is difficult of proof, but, judging from the analogy of cancer in other parts,

it is very probable. For example, a septic ulcer at the side of the tongue due to a carious tooth will become malignant in a few months; on the other hand, chronic inflammatory disease may so mimic malignancy that nothing but a minute microscopical examination can prove its nature. This point will be referred to again under the heading of "Diagnosis."

DURATION OF THE SYMPTOMS.

In cases which eventually come for surgical treatment the symptoms have generally been well marked for many months. In my series, apart from the case preceded by a gastric ulcer, the duration was two, four and a half, eight, nine, twelve, and twelve months, with an average of about eight months. Other writers give similar figures.

It is quite clear, therefore, that in the great majority of cases a most unreasonable and unnecessary delay takes place before surgical means are considered. Three months ought to be ample time in which to carry conviction both to the patient and his doctor that the matter is one which demands radical treatment. Unfortunately, what usually happens is that three months is spent by the patient in vague uneasiness, which he treats by homely or quack remedies; for another three months he places himself under the care of a general practitioner, who goes through a long routine of diet and medicine; then perhaps three more precious months are spent under the observation of a consulting physician, who employs various recondite methods in the diagnosis of his case; and, finally, as a last desperate step, the surgeon is consulted, and it is no wonder that under such circumstances the majority of cases will permit of no curative treatment.

SYMPTOMS, SIGNS, AND DIAGNOSIS.

It seems to me that this part of the subject has been made unnecessarily complicated by undue stress having been laid on exceptional cases and complicated tests. In every one of my series the three cardinal symptoms of pain, vomiting, and rapid emaciation were present. Of these I would regard emaciation as the most significant.

Emaciation.—Makkas [35], in speaking of over 600 cases of cancer of the stomach, states that in not one did this sign fail; and, further, the loss of flesh is of sudden onset, rapid and steady progress. A man with cancer of the stomach will generally lose as much weight in three months as a patient with gastric ulcer does in a year; and, conversely,

the gaining of weight is the only condition which justifies the continuance of palliative treatment in an otherwise doubtful case. It is true that patients with cancer of the pylorus often gain flesh rapidly after a gastro-enterostomy, but I doubt whether this could ever happen as the result of simple dieting. Great significance ought to be attached to the fact that a patient can often tell when the loss of flesh began, and how much it has amounted to, which indicates that a change has taken place of so definite a character as to arrest the patient's attention and make him weigh himself.

Pain is a deceptive symptom here as in most other cases of malignant disease. It is the comparative painlessness of early cancer that is its most dangerous characteristic. Just as a scirrhus of the breast is much less painful than a mastitis of similar duration, or a cancer of the rectum than a fissure of the anus, so cancer in the stomach is, in its early stages, much less distressing to the patient than an ulcer. Probably the early pain is due rather to the coincident dyspepsia than to the growth itself. It is of a dull and heavy character, and is usually much relieved by vomiting. Careful dieting and gastric lavage readily cure the early cancer pain, and this amenability to treatment is another feature which is deceptive as regards diagnosis.

Vomiting.—This sign is of almost constant occurrence when the pylorus is affected, and then it has the well-known features associated with gastric dilatation. That is to say a large quantity of sour-smelling material is brought up once or twice a day. Makkas [35], in 105 cases of pyloric cancer, found that 73 per cent. had vomiting. The percentage was only 53 in those cases where the cancer did not affect the pylorus.

Tumour.—The occurrence and characteristics of a tumour caused by cancer of the stomach present several points for discussion. In my own cases, in only three out of eight was a definite tumour present. In one case a tumour was sometimes absent and sometimes present. In two cases there was a sense of resistance, and in two there was nothing abnormal palpable. Stumpf [46] gives the proportion of operable cases in which a tumour is present as 51 per cent., and Makkas [35] as 79 per cent. (132 out of 167), Mizokuchi [39] as 70 per cent. (43 out of 61). And hence it is quite incorrect to say that any case is too far advanced for radical operation if a tumour is present. A sense of resistance or tumour caused by cancer of the stomach may be due to several distinct factors. In the first place there may be merely a hardening of the rectus muscle over the diseased viscus. Probably this is not so frequent as in cases of gastric ulcer. Then there is the

muscular spasm of the pyloric antrum associated with stenosis. It is this which accounts for the transient or occasional tumour noted in my fifth case. If the patient was given a solid meal, or even if the stomach was merely blown up with air through a stomach-tube, the tumour would certainly make its appearance just under the right costal margin; but when he was kept quietly in bed on light food, not a trace of tumour could be felt. I have elsewhere [18] published another still more remarkable instance of this intermittent tumour. The patient was a young woman, aged 29, who came to the hospital for an epigastric tumour, and because after two days in bed on milk diet it disappeared she refused to have any operation and left the hospital. Within a fortnight she returned with acute dilatation of the stomach and a well-marked tumour of the pylorus. She died within a few hours of readmission; but post-mortem examination showed no gross tumour, although the microscope revealed early cancer of the pylorus with marked muscular hypertrophy. This type of tumour is exactly comparable with that associated with some forms of intussusception. It is not merely a transient hardening but a long-continued tonic contraction. Then there is the tumour caused by the new growth itself. This will be hard, and in thin subjects it is felt to be nodular. Lastly, the tumour may have grown into the parietes or extended into the peritoneum, liver or glands, and this will cause a fixed and well-defined mass of almost hopeless augury. When the tumour is due to a cancer of the pylorus it appears first a little to the right of the mid-line above the navel. It is almost always lower than the normal transpyloric line, because the stomach has undergone dilatation and downward displacement. It is fully movable and much more marked sometimes than at others. As the disease progresses the tumour assumes more definite characters. It is hard and nodular and extends upwards and towards the right, or it becomes fixed to the parietes as a part of an indefinite mass.

Dilatation with marked gurgling and visible peristalsis occurs only in a few cases. This is probably due to the facts that the course of the disease is too rapid to allow of a great degree of muscular hypertrophy, and later in the disease the stomach-wall becomes so infiltrated with cancer that it cannot dilate or contract. The only case in which I have seen very well marked rhythmic peristalsis was my fourth case, in which an ulcer of five years' standing had preceded the cancer; and I am disposed to think that visible gastric peristalsis is always suggestive of a simple stenosis of the pylorus preceding the cancer.

Chemical Changes in the Vomit.—In the majority of advanced cases of cancer of the stomach free hydrochloric acid is absent and lactic and other fatty acids are present; but, as we are considering early rather than advanced cases, these tests are not very much to the point. In my own cases free hydrochloric acid was absent in 5 and lactic acid present in 3 out of 7 in which these signs were noted. Makkas' [35] figures relating to these reactions in 167 cases are:—

Free hydrochloric acid absent, lactic acid present	...	83
" " " " absent	...	20
" " present, " "	...	39
" " " " present	...	14
Not examined	11
		<hr/> 167

Stumpf [46] states that free hydrochloric acid was present in only 3 out of 47 cases submitted to resection. These figures all show that the absence of free hydrochloric acid occurs in the majority of cases, but the all-important point is that this sign is not invariable, and its presence or absence should only be taken in conjunction with other evidence. It is evident that a profound alteration in the gastric secretion will not be likely to take place until the disease is far advanced, and it is just as unreasonable to consider a case to be non-malignant because free hydrochloric acid is present as it would to call any tumour of the breast innocent because no enlargement existed in the axillary glands.

DIAGNOSIS BY INSPECTION AND PALPATION.

Undoubtedly the best and most reliable way of diagnosing a cancer of the stomach is by looking at it and feeling it after opening the abdomen. What chance should we ever have of curing cancer of the tongue or breast if we could only rely upon indirect examination in an early stage? Supposing we never diagnosed a cancer of the tongue until emaciation, fœtor of the breath, and enlargement of the sub-maxillary glands had occurred—would it be possible to submit a single case to radical treatment with any chance of success? In the analogous case of gastric cancer we ought also to insist on the necessity of direct inspection in all doubtful cases. Rapid emaciation, when associated with dyspepsia, should be amply sufficient to demand an exploratory operation. Such an operation in itself involves but little risk, whereas, waiting for the occurrence of a tumour or for the disappearance of free hydrochloric acid involves the enormous risk of the patient being quite

unfit for radical treatment by the time these signs have developed. From this point of view I can hardly conceive there being room for any difference of opinion. But there is another aspect of this question which is more complicated. I refer to the fact that there are many recorded cases in which even the facts discovered by an operation left the diagnosis in doubt. This is well illustrated by my fourth case. A man, aged 55, was in extreme emaciation with well-marked gastric dilatation. On opening the abdomen a nodular thickening occupied the pylorus. A gastro-enterostomy was performed and a piece of the pylorus was removed for examination. The microscope revealed nothing but inflammatory changes (fig. 1). I felt, nevertheless, grave doubt about the matter, and explained it to the patient, who unhesitatingly decided to have the doubtful disease removed. At the second operation the pylorus was much less indurated, and it would have been impossible to point to any gross character of malignancy. Nevertheless it was removed, and a section of its posterior wall showed an early cancer (fig. 2).

The fact is that the outward appearance of an inflammatory and a malignant mass are so similar that it is often impossible to distinguish between them, and in this respect cancer of the stomach is no different from disease elsewhere; and it is a mistake to suppose that a microscopical examination of any chance fragment will settle the matter. Mr. Butlin has insisted on the fact that it is not justifiable to pronounce certain suspicious ulcers of the tongue as non-malignant until they have been cut into serial sections and every section examined; and the case related above proves that this applies equally to growths of the stomach. Still less desirable would it be to depend too much on a hurried histological report made during the course of an operation; but Lund [33] has reported a case in which a positive diagnosis of malignancy was made by the microscope during the operation and in which this was followed by a successful excision at the time.

A most excellent account has just recently been given by Wilson and Maccarty [50] of the relation between ulcer and cancer of the stomach from a histological point of view. These authors founded their observations on 210 specimens of the pylorus excised by the brothers Mayo. Of these, 47 cases were ulcers pure and simple, 5 were ulcers with doubtful evidence of malignancy, 109 were ulcers definitely developing into cancer, 11 were cancers with a doubtful element of preceding ulcer, and 33 were cancers with no evidence of ulcer. So that out of a total of 158 cases of cancer there was evidence of pre-existing ulcer in 125—i.e., in nearly 80 per cent. The illustrations to this paper

are more striking even than the figures. In many cases certain sections are those of simple ulcer, another section from the same case shows irregular glandular proliferation, and yet another shows undoubted cancer. Thus it is convincingly demonstrated that evidence of ulceration not only does not exclude malignancy, but gives strong reason for expecting malignancy in the neighbourhood.

In several cases an area of disease has been removed under the impression that it was malignant, and subsequent sections have shown only inflammatory tissue. Frazier [13] performed a partial gastrectomy for a man aged 54, removing an indurated growth from the pylorus with some enlarged glands. The microscope showed only a simple ulcer. Taylor [47] operated on a woman, aged 60, who had had a three years' history of pain, and in whom free hydrochloric acid was absent. The pylorus was hard and so densely adherent to the rectus that part of the muscle had to be removed with the growth. An excision was done by Kocher's method, and the patient recovered well, but sections showed only inflammatory tissue round the ulcer. In other cases, again, the indurated mass is so hard, fixed, and adherent that the case is treated by gastro-enterostomy only, with the hope of mere palliation, and prolonged survival proves that here, too, the inflammatory mass was wrongly regarded as malignant. Lilienthal [32] operated on a man, aged 40, who had had a hard pyloric growth the size of a duck's egg. He did a posterior gastro-enterostomy as a preliminary to excision, but on re-opening the abdomen sixteen days later the tumour had completely disappeared. Wölfler [51] has related a most dramatic case of this kind. His patient was a young woman whose condition was such that he regarded her as suffering from an inoperable cancer, which could hardly allow her more than one or two months of life. Her emaciation was such that even a palliative operation was thought to be risky; but the unhappy woman fell on her knees and begged that something might be attempted, and accordingly a gastro-enterostomy was performed. Two and a half years later this patient was quite well and leading an active life!

Similar cases of recovery after palliative operation from what was thought to be ineradicable malignant disease are given by the following authors: Paterson [41], a woman alive and well four years later. Kindl [25]: (1) Woman, aged 39; abdomen opened, inoperable tumour found; gastro-enterostomy performed in May, 1905; patient alive and well in May, 1908. (2) Gastro-enterostomy for irremovable growth, January, 1899; nine years later the patient was well. (3) Gastro-

enterostomy, August, 1905, for fixed tumour at the pylorus; good health for over three years, when death occurred in January, 1909, from cancer of the duodenum. In this case it is probable that at the first operation the mass was simply inflammatory, but that later it became malignant. Such a case provides a strong argument for the removal of these doubtful tumours even when the microscope shows only inflammatory tissue.

The disappearance or diminution of a tumour cannot, however, be held to be conclusive proof of its benignity, because in such cases—e.g., as in Case IV. of my series above related—the mass may be composed of much inflammatory and little malignant tissue. The former may disappear and leave the latter as a cancerous germ to fructify later. Dowd [10] related such a case of a young man in which, at a second operation subsequent to gastro-enterostomy, the tumour had apparently gone, and nothing further was done. But eighteen months later the patient died of peritonitis due to the perforation of a malignant growth in the stomach. Krause [27], too, after pointing out that tumours which may have every appearance of malignancy prove to be only inflammatory, observes that after gastro-enterostomy true carcinomata often diminish in size for a time.

Simple ulcers of the stomach may be multiple, and of these one only may be affected by malignancy. Cobb [7] gives an instance of such a condition. In a man, aged 52, a partial gastrectomy was performed successfully for a tumour of the stomach. Subsequent examination proved that there were two ulcers present. One over the pylorus showed early adeno-carcinoma on a chronic ulcer, and the other on the lesser curvature was simple.

Enough has now been said to show the necessity for great care in the distinction between inflammatory and malignant disease of the stomach even after the mass has been exposed by operation. The practical deduction is this: That in all doubtful cases the two-stage operation should be performed and a portion of the tumour taken for microscopical examination at the first stage. If this proves it to be cancer, then doubt is at an end, and resection will follow as a matter of course. If it is inflammatory, then the case must be treated either by excision of the ulcer, or the patient must be seen frequently for twelve months in order to note the first sign of relapse after recovery from the gastro-enterostomy.

OPERATIVE TREATMENT.

Before discussing the value of the two-stage operation, it may be well to review very briefly the various operative methods and their results in order to obtain an idea of the scope that there may be for improvement by this modification. Unfortunately we have in this country no systematic method of recording operations or their results, and we are dependent, therefore, chiefly on the publications from the various Continental clinics. Haberkant [19] in 1896, Makkas [35] in 1907, and Goldschwend [15] in 1909 have given extended statistics from various hospitals in which all the cases are recorded without selection. The last-named author has included in his paper the largest number of cases from other authors who have published their total cases, and it will be useful therefore to summarize his figures whilst referring the reader for details to his original article.

There are three classical operations employed in resection of the stomach. In Billroth's first method an end-to-end junction is effected between the stomach and duodenum after removal of the pylorus. This is without question much the most difficult operation, because it involves adapting a large stomach orifice to a small duodenal opening; and as a consequence of this difficulty many cases die from peritonitis caused by a leakage at the angle of the junction. For example, Paterson [42] refers to a series of 59 cases of gastrectomy by this method, in which 23 per cent. died as the result of leakage, and 12 per cent. more had some leakage without death. In Kocher's method the cut end of the stomach is completely closed and the open end of the duodenum is anastomosed to the back of the stomach. This avoids the A-shaped line of suture, and has given good results in its author's hand, and among recent writers, Ito and Soyesima [21] speak highly of the method. But both these operations (Billroth I and Kocher) are only possible when the disease is of very limited extent, as they both involve a suturing together of the stomach and duodenum. And there must always be a risk of a too meagre removal of tissue in order to avoid tension in anastomosis. In Billroth's second method, after the disease has been removed with a good margin of healthy tissue, both stomach and duodenum are completely closed and the continuity of the alimentary canal is established by a gastro-jejuno-stomy. This method is the one which is now generally adopted. It is applicable to all cases of extensive disease; it allows a free removal of doubtful tissue, and it substitutes a lateral anastomosis for an end-to-

end or end-into-side junction. Makins [34] has shown that in a large series of anastomosis operations on the alimentary canal a fatal leakage takes place in 50 per cent. of the end-to-end junctions and only 30 per cent. in the lateral.

Goldschwend [15] describes the results obtained in 59 cases of gastrectomy by these different methods at the Breslau Clinic thus:—

Method	Number of cases		Died	Percentage mortality
Billroth I	12	...	6	50
Billroth II	34	...	11	33
Kocher	13	...	4	30

and he adds the following statistics from other sources where complete returns are available:—

Method	Number of authors	Number of cases	Deaths	Percentage
Billroth I	11	279	102	36
Billroth II	9	189	77	40
Kocher	3	54	14	26
All methods	19	553	229	51

From these figures it will be seen that radical operations for cancer of the stomach are followed by a very high mortality, varying between 26 and 51 per cent. There are, of course, several series of published cases with a much lower death-rate, especially those of the Mayos [37] (100 cases with 14 deaths), Mayo Robson [38] (100 cases with 13 deaths), and Maydl [36] (100 cases with 16 deaths). But for our purpose it is much more important to mass together the results obtained by a large number of operators than to consider those of the most successful surgeons; because in the one case we are enabled to judge of the method, and in the other case of the man.

It will be observed that in the above table Billroth's second method has a mortality of 40 per cent., whereas Billroth's first method has only 36 per cent., and Kocher's only 26 per cent. This is probably due to the fact that Billroth's second method is used for much more advanced cases, whilst the other two are employed in cases of limited disease. Mizokuchi [39], in a recent paper, reports all cases of resection of the stomach for cancer in Professor Ohnori's hospital. His figures are as follows:—

Method	Number of cases		Deaths	Percentage mortality
Billroth I	5	...	4	80.0
Billroth II	23	...	6	26.0
Kocher	31	...	7	23.5

It must be admitted that the mortality after radical operations for cancer of the stomach is a terribly high one, and the important question is whether any modification of method can be adopted which will improve this state of affairs. The reason for this high death-rate is easy to understand. The patients are often of advanced age. They are reduced to a condition of marasmus by pyloric stenosis. The stomach itself is in a most unhealthy condition because it is the seat of a cancerous mass, and also because its contents are stagnant and decomposing. Is it any wonder, then, that if an extensive resection, followed by an anastomosis, be undertaken that this should result either in fatal shock, or that the patient should have too little recuperative power to resist peritonitis or other post-operative sequel?

I have been able to collect 127 cases in which the cause of death after resection operations is given. They are from the following authors—viz: Goldschwend [15], Rasumowski [44], Müller [40], Branham [3], Frazier [13], Bishop [2], Brunner [4], Garré [14], Krönlein [28], Steinthal [45], Czerny [8], Körte [26], Graham [17], Makkas [35], and Mizokuchi [39]:—

Peritonitis (including "leakage" or "leaking from the duodenal stump")	79 cases
Shock, heart failure, collapse, weakness, marasmus, narcosis	23 ..
Lung complications, especially pneumonia	20 ..
Hæmorrhage	2 ..
Septic cholangitis	1 ..
Brain symptoms	1 ..
Colitis	1 ..

For practical purposes, then, the causes of death are three—peritonitis, shock, and lung complications. It is certain that the danger of all these can be greatly lessened by dividing the operation into two stages. At the first stage a gastro-enterostomy is performed. The patient rapidly gains in strength and vigour, and the stomach is relieved from its stagnation, whilst in many cases the inflammatory element of the tumour mass greatly diminishes. Then, when after a interval of one or two weeks the resection is undertaken, the operation is much shorter and the patient's vitality being greater, there is much less danger of his succumbing either to peritonitis, shock, or lung infection. In my own series of eight cases, in two, the whole operation was performed at one stage, and both these patients died. In six cases the two-stage operation was performed, and of these only one died. I have been able to collect 12 other cases in which the two-stage operation was employed from the following authors: Franke [12] (1 case); Tuholski [49] (2 cases);

Kammerer [23] (1 case); Tixier [48] (2 cases); Jaboulay [22] (1 case); Stumpf [46] (1 case); Mayo Robson [38] (1 case), and three other cases quoted by Mayo Robson [38]. Adding these to my own series, we have 18 cases of which only three died, a mortality of only 16.6 per cent. It may be of interest to give brief abstracts of some of the cases, as it will then be clear that they are often of the most severe type.

TUHOLSKI (CASE I).—This is probably the first instance in which the two-stage method was used. Published in 1890; man aged 30. One year's history of gastric dilatation. Tumour present. Anterior gastro-enterostomy by Senn's method, followed by excision two months later. Died on the same day.

TUHOLSKI (CASE II) IN 1893.—Woman aged 50. History of several months' dilatation of stomach with palpable tumour. Anterior gastro-enterostomy by Senn's method. Excision three months later; good recovery. Died six months later of ptomaine poisoning. No recurrence had occurred, but the anastomosis was reduced to the size of a cedar-wood pencil.

FRANKE'S CASE.—A large adeno-carcinoma involving one-third of the stomach was removed six weeks subsequently to a gastro-enterostomy. The recovery was good.

ROBSON'S CASE.—A man aged 63. Symptoms one year; tumour noticed for one month. Very feeble condition. November 15, 1900: Gastro-enterostomy. A central ring of cancer encircled the stomach. Rapid gain of strength and weight. December 20: The growth was excised. He made a good recovery and lived for sixteen months.

KAMMERER'S CASE.—Man aged 40. General condition was very bad, the hæmoglobin being reduced to 33 per cent., normal. A posterior gastro-enterostomy was done by a Murphy's button. Ten weeks later his condition had greatly improved, the hæmoglobin having risen to 45 per cent. Gastrectomy was then done, but was difficult on account of the adhesions. Recovery was good.

TIXIER (CASE I).—Man aged 59. Extreme cachexia, weighing only 50 kilos. History of dyspepsia, vomiting, and loss of flesh for one year. April 8, 1905: Posterior gastro-enterostomy with a button. June 2, 1905 (two months' interval): Pylorectomy, with removal of a part of the head of the pancreas. Rapid recovery, leaving hospital in thirteen days.

TIXIER (CASE II).—Man aged 37. History of six months' dyspepsia with loss of between 3 kilos and 4 kilos in weight. January 27, 1908: Posterior gastro-enterostomy. His general condition was too feeble to allow more at this time. March 9, 1908: After six weeks' interval, pylorectomy with removal of glands. Good recovery.

JABOULAY'S CASE.—Man aged 58. Two years' history of dyspepsia, no vomiting; loss of 17 kilos. June 15, 1907: Posterior gastro-enterostomy

by button. June 27 (twelve days' interval): Resection of pylorus with a growth the size of an orange. The duodenum was united to the stomach. Good recovery, except for bronchitis and parietal suppuration.

STUMPF'S CASE.—Man aged 64. History of six months' dyspepsia; heart's action irregular, nutrition very bad. Gastro-enterostomy. Later the growth was excised, the part distal to the excision being sewn round a catheter and brought to the surface. Death occurred four days later from pneumonia. There was an abscess in the abdomen round the duodenal fistula.

In addition to these definitely described cases there are many authors who speak well of the method. Such are Quénu [43], Doyen [11], Czerny [8], Hahn [20], Kümmel [29], and Krause [27]. It will be noted that the interval between the two stages of the operation varies very much with different operators. In my own cases the intervals were 7, 7, 10, 12, 12, and 28 days, and I am inclined to think that a fortnight represents the best average time which ought to elapse between the two operations. In cases from other surgeons, however, the interval has usually been much longer, generally about two months—14 days, 6 weeks, 2 months, 10 weeks, 3 months.

It seems to me that such a long interval is quite unnecessary, as patients gain in strength very rapidly after the gastro-enterostomy; and this long postponement gives time for the cancer to spread or to become disseminated, and the adhesions left after the first operation become often densely organized. Kammerer [23], Krause [27], and Tixier [48] in particular call attention to the difficulty caused by these adhesions. But, if the interval is only a fortnight, these adhesions are easily broken down, and they do not add materially to the difficulty of the operation. Robson and Krause both urge against the two-stage method that patients strongly object to undergoing a second operation, and that they may feel so much benefited by the gastro-enterostomy that they will decline to submit to the radical operation later. Of course this is a real difficulty, but I have found that if patients have the matter explained to them, that their best chance of life will be given by having two short operations substituted for one long one, they nearly always give willing consent to this most rational procedure. In one case only (No. VI), that of a very neurotic woman, did the patient elect to have the whole operation performed at once, and she paid the penalty for this choice with her life. It may be quite true that surgeons of great experience and dexterity can perform the whole excision and anastomosis so rapidly as to avoid much danger from shock. But this rapidity is not at all conducive to thoroughness in the removal of outlying glandular tissue, and it is just

as important to patiently dissect out this tissue in cancer of the stomach as it is in the case of the tongue or breast. And, further, the vital resistance of the patient against septic infection is much more lowered by a procedure which lays bare a wide area of tissue at once than by one which allows one absorptive area to heal before another is made.

THE LIMITS OF OPERABILITY.

The varying results obtained by different operators are due in some measure to the different selection of cases for operation. Upon certain general principles all are agreed, but there remain many points about which a difference of opinion must always exist. And yet this subject is one of the deepest importance in connexion with the conversion of the general bulk of practitioners and public to a more favourable view of the surgery of gastric cancer. There are certain operations which every surgeon wishes after the event that he had never attempted. The practical question is whether we can be forewarned of such cases so as to avoid them.

The positive contra-indications for radical operation are: (1) Multiple metastatic growths in the liver or peritoneal cavity; (2) extensive malignant adhesions to neighbouring structures, especially when these involve the portal vein and other great vessels; (3) involvement of distant lymphatic glands—e.g., those over the left clavicle—and (4) great extent of the primary growth, leaving insufficient healthy tissue for anastomosis.

The following factors involve a grave element of doubt: Growth involving a neighbouring organ which may be removable. Such are limited growths in the liver, pancreas, or colon. Müller [40] has described a case in a woman, aged 66, in which he removed a portion of the liver; the patient made a good recovery, but suffered a recurrence in a year's time. There are many cases recorded in which portions of the pancreas have been excised, but the immediate mortality of these is much higher than the average. Childe [5] has related a brilliant success in the case of a woman, aged 35, from whom he removed two-thirds of the stomach, a part of the pancreas, and a segment of the transverse colon at one operation. Makkas [35] gives the best available figures relating to the comparative mortality of these complicated cases. In a series of 81 cases of resection of gastric cancer, 33 died. Of these, in 48 a part of the pancreas was removed and 26 died, and in 9 a part of the colon, of which 5 died. So that we have a total of 81 with an

immediate mortality of 40 per cent., consisting of 57 complicated cases (pancreas and colon involvement), which include no fewer than 31 of the fatalities (54 per cent.), leaving 24 uncomplicated cases with only 2 deaths (8 per cent.).

The mere enlargement of neighbouring lymph-glands does not prove that they are malignant. In my eighth case I removed a number of glands connected with both curvatures of the stomach and from the front of the pancreas, but section showed they were merely inflammatory. In one case related by Makkas the patient's condition was such during a resection that several enlarged glands had to be left, and for this reason early recurrence was anticipated; but she was alive and well three-and-a-half years later. But on the other hand there is the densely hard growth in the glands which mats them together in an unyielding mass quite different from mere enlargement. I would lay special emphasis on this, because I failed to recognize the hopelessness of the condition in my seventh case and was led on to disaster. In this case the growth (shown in figs. 3 and 4) was quite movable and the glands in the curvatures not specially large. But it was in the chains of lymph glands and vessels which run with the coronary and hepatic arteries towards the cœliac axis that the disease had made its furthest extension. And I would make it a rule in future in determining whether a radical removal is possible to tear through the gastro-hepatic omentum and carefully examine the region occupied by the branches of the cœliac axis. If these are obscured by a hard nodular growth, it is very difficult to secure the arteries and veins, and it is impossible to remove all the disease which runs up by the side of the aorta. Glands which lie firmly embedded in the pancreas can be removed with a part of that organ, but it is the glands above the pancreas, fixed to the hepatic, splenic or coronary vessels, which form a contra-indication to radical operation.

Makkas [35] gives a list of 128 cases of cancer of the stomach in which the presence of apparently malignant glands was noted. His list is as follows:—

No glands apparent	14
Glands in small curvature alone	39
" large	"	"	13
" both curvatures	51
" " and in pancreas	17
						<hr/> 128

Whatever general rules are laid down for the selection of cases suitable for operation and for excision, individual surgeons will always differ

very much in what they consider may and ought to be attempted. This is well seen from the following list from Kausch [24], who has collected large lists from four different sources :—

Operators	Total number of cases	Number operated upon, including palliative or exploratory	Percentage	Resection	Percentage
Boas ...	234	48	20	11	4.7
Krönlein Schonholzer }	264	197	75	50	19.0
Mikulicz Hoffmann }	665	458	69	164	24.7
Körte Nordmann }	126	115	91	38	30.0

There is a very important point which bears upon this question of radical operability in connexion with the two-stage method. It has already been shown how very rapidly a mass which is partly malignant and partly inflammatory diminishes in size after a gastro-enterostomy. If, therefore, at the first operation the mass appears to be very hard and fixed, it is always worth while to simply do the gastro-enterostomy and re-open the abdomen in a month's time, when the inflammatory fixation will be found to have much diminished.

THE REMOVAL OF THE LYMPH AREA.

As regards the extent of the radical operation, I wish to speak chiefly about the removal of the lymphatics. A pretty general agreement exists concerning the amount of the stomach and duodenum which should be removed. This consists of the whole of the lesser curvature or at least that part of it up to the point where the coronary artery and lymphatics join it; about a half or a third of the greater curvature and about 1 in. of the duodenum. But in connexion with the removal of the lymphatic area associated with the stomach there has been hitherto no definite plan at all comparable, for example, with that adopted in cancer of the breast. So far, all that has been done and advised is the removal of the lymph-glands connected with the stomach along its lesser and greater curves, together with certain glands on the anterior face of the pancreas. But it is obvious that this by no means includes all the lymph paths which lie in the path of what Sampson Handley calls lymphatic permeation. The largest lymph-bearing area connected with the stomach is the great omentum.

Before discussing the possibility of the removal of this wide area of peritoneum, it may be well to give some evidence that cancer-cells do

really travel thus along this tissue. Fig. 7 is the drawing of a slice taken from the tissues of an old woman who came into the General Hospital with enormous ascites, and who died very soon after this had been tapped. She had an advanced colloid carcinoma of the stomach which had spread widely in the peritoneal tissues. This diffuse growth had spread itself in no disorderly fashion, but in the definite planes provided by the lymphatic areas of the omenta. Thus it will be seen that a continuous mass can be traced from the stomach, up the gastro-hepatic omentum, and down the great omentum, when it turns back in front of the colon which is not involved, along the upper surface of the transverse meso-colon to the tissues in front of the pancreas. Further, the



FIG. 7.

A slice taken in a sagittal direction from the abdominal viscera in a case of colloid cancer of the stomach. A, great omentum; B, primary growth at the lesser gastric curve; C, small omentum; D, duodenum; E, pancreas; F, transverse meso-colon; G, colon. Note that exactly those areas indicated in fig. 8 are here infected with cancer. (Half natural size.)

free margin of the great omentum was adherent in the pelvis, and from this point an exuberant mass of growth extended over the whole of the pelvic floor. It is clear, then, that in this case the whole of the great omentum had become permeated with the cancer, and had served as a carrier of cancer-cells to the pelvis.

It may be objected that, as this is a case of such advanced disease, no inference can be drawn from it relating to the early stages of

cancer dissemination. But I have been able to demonstrate the same process in a much earlier stage of the disease. The parts removed from my seventh case are shown in figs. 3 and 4; but a part of the great omentum has been cut off for purposes of microscopical examination. On beginning the removal of this mass one week after a posterior gastro-enterostomy, I was surprised to find that the edge of the great omentum was adherent somewhere in the right iliac fossa. This adhesion had not been noticed at the time of the first operation. A very little manipulation served to separate the adherent parts, and it was noted that

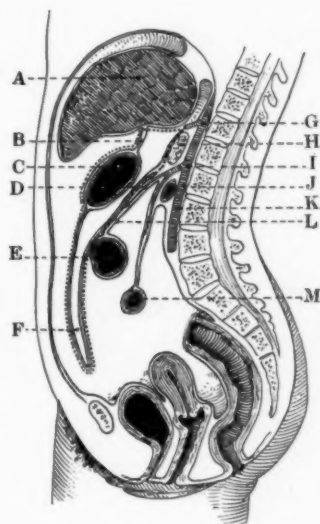


FIG. 8.

Diagram of peritoneal relations as seen in sagittal section. The dotted line surrounds the parts removed in the proposed operation. A, liver; B, small omentum; C, stomach; D, middle colic artery; E, transverse colon; F, great omentum; G, colic axis; H, pancreas; I, superior mesenteric artery; J, duodenum; K, aorta; L, transverse mesocolon; M, small intestine.

the point to which the omentum was fixed was at the root of the mesentery, adjacent to the ileo-cæcal region. The actual point of the omental edge thus separated was apparently thickened and inflamed. After the patient's death the point on the mesentery from which the omentum was separated could be identified as an injected area. Both these tissues (the tip of the omentum and the mesentery) showed

unmistakable cancerous infiltration, as is seen in the figs. 5 and 6. And this malignant growth at the extremity of the omentum, invading the tissues at the pelvic brim, existed with an omentum which otherwise appeared perfectly free from disease. I would venture, therefore, to assert that these cases, confirming as they do what we should expect of the lymphatic areas connected with stomach cancer, prove that the whole of the great omentum ought to be removed in every radical operation.

In confirmation of this suggested method by which cancer spreads from the stomach through the great omentum to the pelvic organs, it may be noted that among the scanty information we possess as to the condition of patients dying from recurrence of the growth after operation there are several instances of the recurrence taking place in the pelvis. Goldschwend [15] mentions the case of a patient who died, forty-one months after resection of the stomach, of malignant disease of the ovary. Makkas [35] relates two cases—one in a woman aged 39, the other in a woman aged 40—who died of recurrence in the ovary or pelvis. Goullioud [16] had a female patient who died, seventeen months after partial gastrectomy, of cancer of the ovary.

Dobson and Jamieson [9] have recently made preparations showing the lymphatics connected with the stomach. I have shown the chief glands groups which they describe in fig. 9. They state that the glands associated with the hepatic artery lie along the upper border of the pancreas, whilst a chain of four to seven glands is associated with the right gastro-epiploic artery lying below the vessel and having a tendency to stray down between the layers of the great omentum, especially in the case of adults. These never extend to the left of the middle of the great curve, and their efferent vessels all end in the sub-pyloric glands. It is also noted that some of the lymph-vessels from the pylorus are not connected with the lower coronary glands, but go direct to the glands that lie below the origin of the coronary artery. Lymph-vessels from the great curvature run into the great omentum, and, finally looping back, end in the gastro-epiploic groups. Lengemann [30] found in cases of gastric cancer the following involvement of glands: coronary in 50 per cent., glands on the great curve 37 per cent., and sub-pyloric groups in 60 per cent.

If it is admitted that the great omentum ought to be removed, this is easily carried out by cutting through the peritoneum, which passes from the back of the omentum to the front of the transverse colon; and in many cases it is then possible to strip the peritoneum off the upper

surface of the transverse meso-colon to the front of the pancreas. This is shown after removal in fig. 4.

The complete removal of the small omentum is fortunately not so important, because it has far fewer lymph-vessels than the great

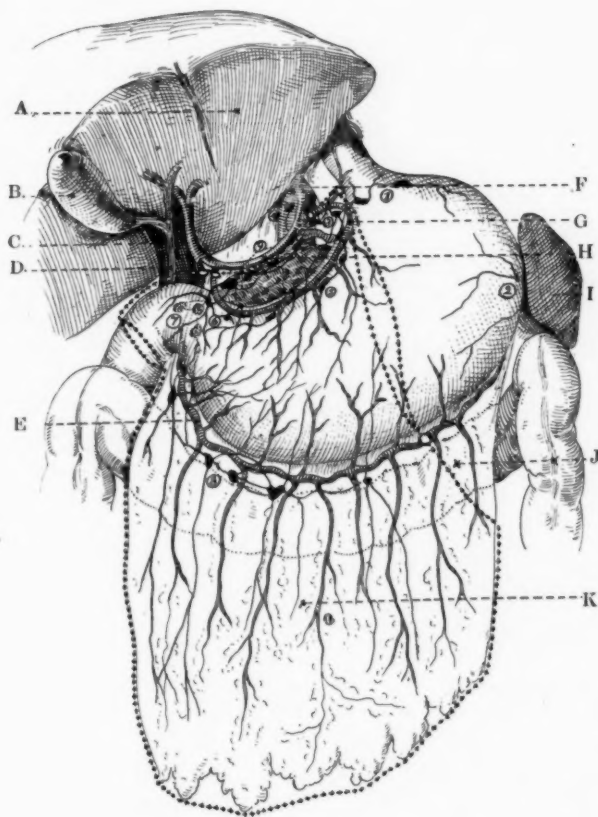


FIG. 9.

Showing the lymph glands and vessels of the stomach and the lines of the proposed excision. A, liver; B, gall-bladder; C, portal vein; D, bile-duct; E, gastro-epiploic artery; F, aorta with coeliac axis; G, coronary artery; H, hepatic artery; I, spleen; J, colon; K, great omentum. Numerals represent lymph glands: 1, paracardial; 2, splenic; 3, upper coronary; 4, lumbar; 5, lower coronary; 6, supra-pyloric; 7, sub-pyloric (these ought to have been placed lower down in the angle between the pylorus and first part of the duodenum); 8, gastro-epiploic; 9, supra-pancreatic. The dotted line surrounds the area to be removed.

omentum. It cannot, of course, be entirely removed, because it is wrapped round the portal vein, bile-duct, and hepatic artery. The lymph-vessels from the upper margin of the stomach run in company with the gastro-duodenal and hepatic arteries on the one side and with the coronary artery on the other, behind the peritoneum, which covers the pancreas at the back of the lesser sac of peritoneum. This is the same layer of peritoneum which is continuous with the posterior layer of the great omentum, and it may be removed with it. The coronary artery should be tied as it comes off from the cœliac axis; the gastro-duodenal artery as it comes off from the hepatic.

The operation which I have performed for the complete removal of the lymphatic area connected with the stomach may then be described as follows (*see* figs. 8, 9, 10):—

(1) *Posterior Gastro-enterostomy*.—The anastomosis is made as far towards the left as possible, certainly to the left of the middle colic artery. The abdominal wound is closed and the further stage is performed in about ten days' time.

(2) *Division of the Gastro-hepatic Omentum*.—This can usually be done without any bleeding. The line of division should be close to the liver, from the œsophagus to the hepatic vessels, and then vertically down the mesial side of the latter.

(3) *Ligature of the Coronary Artery*.—The posterior wall of the lesser sac of peritoneum is now exposed and two folds of peritoneum are seen to run towards the left and right from the upper border of the pancreas. These are the left and right pancreatico-gastric folds, and they contain the coronary and hepatic arteries respectively. The peritoneum is divided above the origin of the coronary artery, and this vessel, with the companion vein and lymphatics, is ligatured and cut close to the cœliac axis. The peritoneal incision is continued along the upper border of the coronary vessels to the lesser curve of the stomach, following the line of the left pancreatico-gastric fold.

(4) *Ligature of Branches of the Hepatic Artery*.—The division of the peritoneum at the back of the lesser sac is continued towards the right just below the hepatic artery along the right pancreatico-gastric fold. The pyloric and gastro-duodenal vessels are ligatured close to the hepatic artery.

(5) *Freeing of the Great Omentum*.—The point on the greater curvature of the stomach where the resection is to be made having been determined, the omentum is divided from this point outwards towards its left free margin, the terminal part of the left gastro-epiploic artery

being ligatured. The great omentum is then turned up and separated from the anterior surface of the transverse colon. This is easily effected by blunt dissection aided by gauze stripping.

(6) *Division of the Stomach and Duodenum.*—This is carried out between two pairs of forceps in each situation and calls for no special comment.

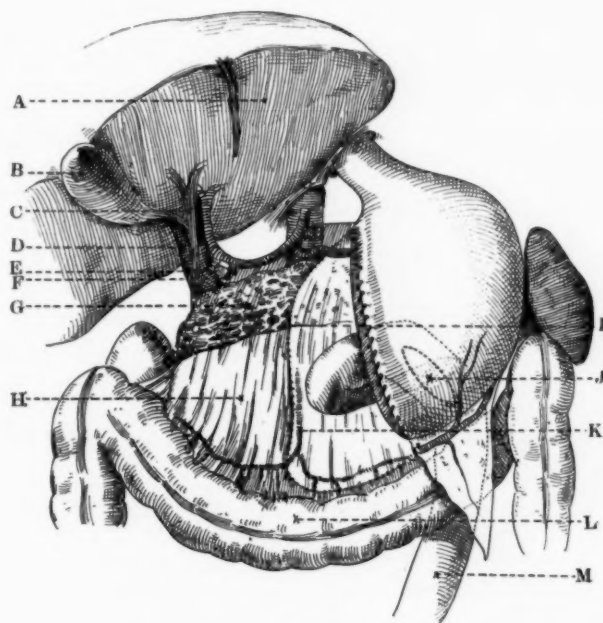


FIG. 10.

Parts after the completion of the proposed operation. A, liver; B, gall-bladder; C, bile-duct; D, portal vein; E, stump of gastro-duodenal artery; F, stump of pyloric artery; G, pancreas; H, transverse mesocolon, from which the base of the great omentum has been partly stripped; I, cut edge of peritoneum lining the lesser peritoneal sac; J, gastro-jejunostomy opening; K, middle colic artery; L, transverse colon; M, jejunum. This ought to be compared with fig. 4, which represents the parts actually removed.

(7) *Separation of the Pre-pancreatic Peritoneum.*—The pylorus remains now attached only by the peritoneum passing from its posterior surface and lower border to the front of the pancreas. This can be stripped off by gentle gauze pressure, the base of the great omentum

coming away from the transverse meso-colon. If there is evidence of macroscopic disease in the glands round the head of the pancreas, it is better to take away a thin slice of the viscus. The separation of the root of the omentum from the meso-colon should only be carried as far as the middle colic artery and vein, for fear of injuring these structures. If the whole mass be now lifted up it is attached only by peritoneum to a line running from the œsophagus to the middle colic vessels. This is cut through, and the excision is complete.

(8) *Sewing over the Cut End of the Stomach and Duodenum.*

(9) *Hæmostasis and Closure of the Wound.*

By this operation the entire lymphatic area connected with the pyloric end of the stomach is removed in one piece. It may be objected that it is not possible to remove the great omentum without injury of the colon and its blood-supply; but if there is no actual infiltration of these structures it is quite easy to carry out. In fig. 8 there is represented the relation of these parts in early foetal life. The great omentum is really the dorsal mesentery of the stomach, and has an attachment to the posterior abdominal wall which is quite independent of the transverse meso-colon, which becomes adherent to it in later life; and even in the adult it is quite easy to show that the omentum merely passes over the front of the colon and its mesentery; and in the case I have figured in fig. 7 is seen an instance where the malignant extension follows the same path, leaving the colon untouched. Figs. 3 and 4 represent part of the stomach with the omentum removed by this method. In the posterior view the peritoneum, passing from the omentum over the head of the pancreas, is well seen.

If there is any infiltration or if there are actually malignant glands at the base of the omentum where it joins the meso-colon, then, of course, the matter is different. The removal of a wide extent of the transverse colon with its meso-colon then becomes necessary.

But I am speaking chiefly of the routine procedure to be adopted in cases of early disease before any obvious lymph metastasis has taken place. The proportion of recurrences in successful cases of resection is so high that one is forced to the conclusion that the removal is not sufficiently radical. Now everyone will admit that if a cancer of the bowel is resected, however early be the condition, its mesentery should be removed as far as possible; and my contention is that the great omentum is in reality the dorsal mesentery of the stomach, and it should therefore be removed, together with its peritoneal extension in front of the pancreas right up to the root of the cœliac axis.

REMOTE RESULTS OF RADICAL OPERATION.

In the estimation of the remote results of these operations there are many difficulties, such as the patients being lost sight of, and so forth. It may be presumed that a certain proportion of patients of whom no news can be obtained do survive for three years or more. On the other hand, a certain number of those who are well three years after the operation die of recurrence at a later date. These two sources of fallacy tend to counterbalance one another, and we may therefore gain an approximate idea of the expectation of life from the large series of figures published in the German clinics.

In the two following tables the cases alive and well at the end of three years are compared with (1) the total number subjected to resection, and (2) the number of cases which survive operation.

TABLE SHOWING PROPORTION OF THREE-YEAR SURVIVALS TO NUMBER OPERATED UPON.

Author	Total number of cases resected	Cases alive and well three or more years after resection	Percentage
Goldschwend [15] ...	59	7	12.0
Clairmont [6] ...	498	30	6.0
Makkas [35] ...	163	17	10.0
Kindl [25] ...	16	2	12.5
Total ...	736	56	7.6

TABLE SHOWING PROPORTION OF THREE-YEAR SURVIVALS TO THE TOTAL NUMBER RECOVERING FROM OPERATION.

Author	Total number of cases recovering from resection	Cases alive and well three or more years after resection	Percentage
Goldschwend [15] ...	38	7	18.0
von Eiselberg [15] ...	26	3	11.5
Mikulicz [15] ...	63	4	6.3
Caspersohn [15] ...	9	4	44.0
Makkas [35] ...	92	17	18.4
Kindl [25] ...	9	2	—
Total ...	237	37	15.6

It will be seen from these figures that the proportion of those operated upon who survive more than three years is about 7 per cent., whilst the proportion of those recovering from operation who thus survive is about 15 per cent. Such results evidently show that there is great room for improvement in the thoroughness with which the radical operations are performed, and it may be that such improvement lies in the direction which I have above indicated.

There are many records of long survival after resection of the stomach for cancer. Goldschwend [15] refers to cases alive and well four-and-a-half, five, and seven years after; Makkas [35] to 4 cases four years, 5 cases five years, 3 cases six years, 1 case ten years, and 1 case twelve years after. Leriche [31] was able to collect 146 cases surviving over three years, one of these being sixteen years, 8 ten years, and 48 five years after resection.

As regards the average length of life of those dying of recurrence, there is a quite remarkable unanimity among different observers. This time is given by various authors as follows:—

Goldschwend [15]	15 months, 20 days
Kausch [24]	18·3 „
Kocher [24]	18·7 „
Krönlein [28 and 24]	18 „

As the average length of survival after simple gastro-enterostomy for cancer is about six months (six months and ten days was the average in Goldschwend's list of 106 cases), it may be claimed that the radical operation, even when it does not prevent recurrence, prolongs life for a year longer than a merely palliative operation.

In conclusion, the various opinions discussed above may be summarized as follows:—

- (1) That a quite unreasonable period is usually allowed to elapse before the patient is presented for surgical treatment.
- (2) That in the majority of cases the diagnosis is to be made from the cardinal symptoms of loss of weight, vomiting, and pain.
- (3) That all doubtful cases should be submitted to exploratory operation unless body-weight rises under medical treatment.
- (4) That even exploratory operations often leave the diagnosis doubtful. In such cases the case should be seen at frequent intervals, or else treated by excision.
- (5) Mortality after resection of cancer of the stomach is between 26 and 50 per cent. This is caused by peritonitis, shock, and lung complications.
- (6) The immediate mortality can be greatly reduced by adopting the two-stage method.
- (7) That the mortality can be further much reduced by excluding cases where the colon, pancreas, or posterior lymph-glands are seriously involved.
- (8) That the adequate removal of the associated lymphatic areas demands that the great omentum and the tissues in front of the pancreas should be taken away.

(9) This can be done by the above described operation.

(10) Remote results of radical operations only give a percentage of 7.6 three-year recoveries of those operated upon. This low figure may in part be due to the inadequate removal of lymphatic tissues.

In conclusion, I must express my great indebtedness to Mr. Stuart V. Stock for his kindness in making all the lantern slides which have illustrated this paper.

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158 Groves: *Radical Operation for Cancer of Pylorus*

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DISCUSSION.

The PRESIDENT (Mr. Rickman J. Godlee) thanked Mr. Groves for his interesting contribution, which raised two particular questions: first, if it was advisable to do the operation of pylorotomy in two stages; and, secondly, as to the advisability or possibility of taking away the great omentum from the anterior surface of the transverse colon.

Mr. PATERSON said the Section was much indebted to Mr. Groves for coming up from Bristol in order to give them such an interesting and encyclopædic paper on the subject of cancer of the stomach. In these days, when surgery had reached such a degree of perfection that all removable organs could be removed, irremovable ones stitched, and those not amenable to such treatment incised or partly resected, it required considerable ingenuity to discover a new method of operation, and Mr. Groves was to be congratulated on having brought one forward that evening. Time would allow him to allude to only two points in the paper. One of the most important concerned the performance of partial gastrectomy in two stages. He thought the author was unduly pessimistic as to the results, so far as immediate mortality was concerned, of the operation done in one stage. He had collected the statistics of Kocher and the Mayos, and several other surgeons, at the time that he delivered his Hunterian Lectures, and the average mortality, at the hands of a number of surgeons, was under 20 per cent. And when he was in America, in 1907, the Mayos told him they had performed over 100 resections of the stomach with a mortality of exactly 14 per cent. Mr. Groves gave a table showing the causes of death after resection of the stomach. According to this table, 79 per cent. of the cases which died did so owing to peritonitis, either from leakage or other causes. In the present stage of surgical technique that was a result which no self-respecting surgeon would dream of; or, if he did, he ought not to attempt resections of the stomach. He did not think the mortality of the operation in two stages was as low as Mr. Groves maintained; he (Mr. Paterson) knew of several published cases which Mr. Groves had not included in his table. Mr. Paterson had recorded one case himself which he

did in two stages, and that patient, unfortunately, died. One would rather expect the second operation to present difficulties, because the adhesions which formed as the result of the first operation complicated the operation which was performed at a later stage. And in some cases the manipulation incidental to resection of the stomach had been sufficient to separate the recently-performed anastomosis, so that at the second operation the whole procedure had to be done over again. Yet, notwithstanding these drawbacks, he had come round to the opinion that, on the whole, the operation in two stages was perhaps best, considering all the circumstances. One was met with a very difficult problem in treating cancer of the stomach, and it was necessary to decide, taking many circumstances into consideration, what was the best method of procedure. He thought, therefore, it was the best plan, at any rate in advanced cases, to do a gastro-jejunostomy first, and then feed up the patient for two or three weeks, so that he might be in a better position to undergo the resection of the stomach. There had been many objections urged against that. Jacobson, in his recent edition, objected that the subsequent resection of the stomach would disturb the previously-formed anastomosis, and so cause complications; and that was what happened in the first case which he (Mr. Paterson) did. This, however, was a complication which might be avoided. With regard to the statistics given by Mr. Groves as to the period of survival after operation, there again he was unnecessarily pessimistic. He remembered that at least 21 per cent. of Kocher's cases were well three years after the operation, and he believed that just over 11 per cent. were well five years after the operation. It was rather difficult to criticize Mr. Groves's suggestion as to operative treatment, because he was rather in the dark as to the difficulty of the method; but he felt that, as a rule, patients with carcinoma of the stomach were so enfeebled that anything which added to the severity of the operation must be most carefully considered. He confessed he was not inclined to add to resection the procedure of peeling off the omentum, though he agreed as to the risk of the cancer spreading down the omentum after the stomach had been removed. He lost one of his cases three and three-quarter years after operation from that cause. Then post mortem it was found that a huge mass which the patient had had for six months was not cancer, but an inflammatory mass round a stercorous ulcer, caused by a very small growth in the colon or omentum, which had caused an annular stricture of the colon and led to an accumulation of faeces behind the stricture. In that case, if the omentum, or, better, the transverse colon, had been removed, possibly recurrence would have been prevented. He wished to dissent most emphatically from one opinion apparently held by Mr. Groves. Mr. Groves stated that in doubtful cases of gastric carcinoma an exploratory operation should be performed *unless* the patient's body weight improved under medical treatment. In other words, Mr. Groves held that increase of body weight negated the presence of cancer. He believed that view to be entirely erroneous. A patient suffering from carcinoma of the stomach frequently gained considerably in weight as a result of careful dieting and rest in bed, and he had known patients gain two or three pounds in weight in ten days as the

result of such treatment. It could not be known too widely that the presence of a cancerous growth was compatible with gain in weight. He was loth to criticize Mr. Groves's plea for a more radical operation, because he felt so strongly that the more radical the operation the better the ultimate results. If he did adopt a more radical operation, he would prefer to resect not only the omentum but the transverse colon with it, an operation which he had performed on one occasion; but at present he felt doubtful as to how far such an extensive operation was justifiable, considering the condition of most of the patients when they came under the care of the surgeon. His opinion was that future success in operating for gastric carcinoma must depend, not on adding to the extent of the operative procedure adopted, but on earlier diagnosis.

Mr. W. G. SPENCER said he was sorry Mr. Groves and Mr. Paterson had not approached the important point as to the danger of submitting the patients to resection of the stomach. German statistics were quoted, and they might accept them as either 15 per cent. or $7\frac{1}{2}$ per cent., or, referring to a few cases of Professor Kocher, there was a survival till five years afterwards of 11 per cent. But neither English nor American statistics had been quoted, in which, as far as he knew, there were only single cases of survivals over three years from operation, and certainly over five years. He (Mr. Spencer) had had a series of cases, especially from Dr. de Havilland Hall and Dr. Murrell, in connexion with the Westminster Hospital, from 1895, and those physicians had diagnosed and carefully selected the cases, but very few of them could reasonably be submitted to resection. Either the cases were far too advanced and gastro-jejunostomy had relieved them for a short time, or they had been the early cases in regard to which he still felt in doubt as to whether they were carcinoma or not. Even in the case which Mr. Groves quoted and showed on the screen, he thought some pathologists would cavil at the idea that it was cancer, and might regard it as merely some inflammatory proliferation of the numerous glands. Mr. Paterson had pointed out the danger of the two-stage operation; and even if it were correct to carry out the procedure, the mere fact of going up to the origin of the gastric artery, and down to and including the omentum, increased the gravity of the operation. As an alternative, there was gastro-jejunostomy. There might, in exceptional cases, be some complication, but with regard to that operation one might say that the operative mortality had been reduced to almost *nil*. Certainly the survivals might be surprising, because in several of the cases he was sure he had done gastro-enterostomy for extensive carcinoma, but the cases being alive and well a long time afterwards, he must have been mistaken. There had not only been the nodular mass, but glands which felt carcinomatous. The last case, which he resected six months ago, seemed to be positively an annular, hard, nodular mass at the pylorus, which it seemed rational to excise, with a deep excavation running down to the head of the pancreas. He had done an extensive excision, but the patient got well, and the pathologist reported that there was no sign of carcinoma—that there was only inflammatory tissue. Therefore he felt great difficulty in following the proposal of Mr. Paterson and Mr. Groves, especially in such

doubtful cases. It might be that the Germans had more slow-growing forms of carcinoma and a more favourable type for operating upon than we had in England, for here in comparatively few could one be sure that excision was the best thing to do.

Mr. JONATHAN HUTCHINSON desired to endorse the views which had been expressed by Mr. Spencer, and he suggested that some of the statistics reported from Germany might have erred, quite innocently, in that cases of inflammatory thickening of the stomach with doubtful pathological changes had been recorded as cancer. Such might be those of Billroth, in which the patient was alive twelve years after excision of cancer of the stomach. It was fair to say that, because those present must have had, like himself, experience of excision of a thickened and nodular pylorus with enlarged glands, where only the examination of slide after slide by an experienced pathologist had decided the question. He was sure cases in the German statistics had been accepted as cured three years after operation where the evidence was not sufficient to justify their being regarded as cancer at all. He also endorsed Mr. Spencer's doubt that the case of which Mr. Groves showed a slide was cancer. In that particular slide at least the appearances were against cancer. He asked whether, in the six cases, Mr. Groves found the difficulty which one would have anticipated in excising the stomach from the position of the gastro-jejunostomy aperture. He would be surprised to hear that Mr. Groves did not meet with such difficulties. He (Mr. Hutchinson) was sceptical about the value of excising the great omentum. Knowing how closely it was adherent to the colon, and how, therefore, that procedure would add to the difficulty of the operation, he doubted whether it increased the chances of the patient surviving the operation for long.

Mr. C. A. BALLANCE remarked that, after the sceptical remarks of Mr. Spencer and Mr. Hutchinson, he would like to say a few words. He thought the Section was much indebted to Mr. Groves for having brought forward, his contribution in so clear and admirable a manner, and he was sorry others had not been present to listen to it. The paper threw him back many years, when, in 1884, he saw Professor Thiersch remove a pylorus in Leipzig for the first time. He had been practising it with his two assistants. The operation of resection of the pylorus and the union of the cut end to the stomach and duodenum was completed in little over an hour. At the end of six weeks he saw the patient again, and she was then eating a mutton chop. He did not know how long she lived. On his return to London he was present at a similar operation. The same care had not been taken in preparing for it, and it took nearly three and a half hours; and the result was that the patient died shortly after the operation. Of course since then much knowledge had been gleaned about gastrostomy and gastro-enterostomy. He thought Mr. Groves was particularly to be congratulated upon his study of the lymphatics in the region of the stomach. He appeared to have attempted to do for the stomach what Halsted did for the breast. He had pointed out that if there was to be more success at operations on the stomach for cancer of the pylorus it would be necessary to

make a much wider removal of the peritoneal structures around the pylorus than was customary at the present time. He therefore thought Mr. Groves's paper led in the right direction. One could not know much about cases of pyloric tumour until one had opened the abdomen; he did not think anyone could promise to remove a pylorus until he had seen and handled it. Only the previous day he opened an abdomen with the intention of removing the pylorus, but he found the disease much too extensive for such an operation. Four months ago he operated upon a case which was very interesting to him; it was of the type which had been referred to in the paper. The man had been through the South African war and had suffered great hardships. For five years he had had very severe stomach symptoms, and for three months he had been daily washing out his stomach. He was greatly emaciated. Mr. Ballance was unable to find any tumour by palpation without giving chloroform. The stomach was somewhat enlarged, and he found a hard lump involving the upper part of the pylorus and extending a considerable way along the lesser curvature. He felt no doubt it was carcinoma of the pylorus, and that it had arisen in the chronic inflammation of the stomach. He (Mr. Ballance) did the two-stage operation, which he regarded as by far the best, and in the cases in which he did it he had remarked that after a gastro-enterostomy the removal of the pylorus did not produce much shock. Therefore in every pylorectomy he would advise a gastro-enterostomy first, and at a second stage the pylorectomy. He had no experience of the difficulty of the two-stage operation, but he did not think there was much, nor any risk of displacement or injury of the union between the jejunum and the stomach. In the case referred to he did a gastro-enterostomy, and, ten days later, pylorectomy, and there was no shock at all. After the pylorectomy there was a little difficulty in taking food for some time, and he thought the reason was that the anterior support of the stomach was lost, and the altered relations of the gastro-enterostomy opening. The main point about the case was the examination of the hard, nodular lump which he had removed with the pylorus. Mr. Shattock examined it, but could not find any carcinomatous tissue in it. Mr. Shattock had seen only two other cases like it; one of them was given to the College by Mr. Mayo Robson, and the other was presented by Mr. Makins. Mr. Shattock did not make serial sections of it, and Mr. Ballance said to him that unless he did that he would still feel doubtful as to it being an innocent lump. On the epithelial aspect of the tumour, which occupied all the coats of the stomach except the epithelial, there was an area the size of three pins' heads where the epithelium was absent, so that that might be looked upon as an ulcer. He thought the case would probably get well and that there would be no recurrence; but that kind of case could not be instanced as one in which successful pylorectomies had been performed for cancer. There were other cases, such as the President and others must have seen, in which the abdomen had been opened with the intention of doing gastro-enterostomy or some other operation, but the disease was found to be so extensive that nothing had been done and the abdomen had been closed. He had known three cases of that kind—one involving the stomach, and two others

the large intestine—and in each of them the patient had got quite well, and subsequent examination at later periods showed that they had remained well. There seemed to be some strange condition, very rarely present, which caused what apparently was malignant disease to disappear when the abdomen was opened. In conclusion, he desired to thank again Mr. Groves for his most admirable paper.

The PRESIDENT said he would like to add to the questions which had been asked, two of his own—one physiological, and one anatomical. Had it been proved that there was a circulation downwards in the anterior layer of the great omentum and upwards in the posterior layer? Also, was it really possible to peel off the great omentum from the anterior surface of the transverse colon, and the upper surface of the transverse meso-colon, and leave the serous surfaces behind, or, when the great omentum was peeled off, was a raw surface left? And, after the peeling, did one expose the branches of the middle colic artery?

Mr. GROVES, in reply, said Mr. Paterson thought he had been too pessimistic about the mortality; but he considered he had taken the only view possible, for, apart from collecting the statistics from the various sources, he had not expressed a personal view. He had a passage in the paper, but did not read it, about the difficulty brought about by adhesions. That was a matter concerning the interval between the two operations. The shortest interval he had had was seven days, and the longest twenty-eight days. Any interval between a week and a fortnight made no difference in the difficulty of the second operation, as the adhesions were so light that they were a negligible quantity. And the patient was much better, and one felt that one had a comparatively straightforward task instead of a long and hazardous one. He did two cases at one sitting, and both died; six others he did at two sittings, and all except one got well. With regard to removal of the great omentum, he could positively assert that the operation was not a bit longer or more difficult on account of removing the whole of the great omentum—i.e., all except the left-hand corner where the great omentum joined the gastro-splenic omentum. And he did not think it took as long as the operation of tying off the great omentum after ligature of both ends of the gastro-epiploic arcade. There were many blood-vessels running from the gastro-epiploic channels to the curvature of the stomach, but when separating the great omentum from the colon one was separating a comparatively avascular area. Anyone who tried it in the dissecting room or on the operating table would have no difficulty in removing the great omentum from the front of the transverse colon. He spoke with more diffidence about removing the peritoneum from the base of the great omentum and from the head of the pancreas, and the President mentioned injury to the transverse meso-colon. In the two cases in which he did it during life he had no difficulty in stripping the peritoneum from the front of the transverse meso-colon, but he kept to the right of the middle colic artery. There was a strip over the front of the head of the pancreas which contained a number of

lymphatic vessels. He was much amazed to hear Mr. Spencer and Mr. Hutchinson refer to the statistics he had put forward as if they were rosy; he himself regarded them as gloomy. He would put Mr. Paterson's idea that he (Mr. Groves) took a pessimistic view against Mr. Hutchinson's and Mr. Spencer's view that it was too optimistic. He had not really taken any view, but had given the best available statistics. He knew of no statistics where there was a less margin allowed for error than the German. He agreed that there was room for doubt as to whether the slide which had been discussed was that of cancer. But he had asked two independent professional pathologists to report, and they both said there was no doubt about its nature. Perhaps in the microphotograph the most instructive piece of tissue had not been chosen. In his third case he had made the opening of the gastro-enterostomy to the right of the middle colic artery, and he had to be content with a very much smaller pylorotomy than otherwise. And, he feared as a result of it, his patient died in eighteen months, whereas, if he had kept to the left, he might possibly have been alive now. He was interested to hear Mr. Ballance say, in his kind remarks, that provided the second operation were done within ten days of the first, there would be no difficulty in carrying it out. In answer to the President's first question, he did not know what physiological proof would be possible, but Prussian blue could be injected from the stomach down the great omentum. That was the only evidence, and it was quoted chiefly from Dobson and Jamieson.¹

¹ I have, since this discussion took place, had the advantage of a personal communication from Dr. Jamieson. He states that he cannot find any vessel in the great omentum which is not taken up by the gastro-epiploic, sub-pyloric, or splenic glands. In Dobson and Jamieson's paper the fact is stated that the lymph-vessels run downwards into the great omentum, also that the efferent vessels from the sub-pyloric glands run downwards over the head of the pancreas, and others run upwards over the body of the gland to the supra-pancreatic group. It is such vessels I would seek to remove with the peritoneum on the front of the pancreas.

Surgical Section.

March 8, 1910.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

Rupture of the Tunica Vaginalis in Hydroceles.

By SOMERVILLE HASTINGS, M.S.

It would appear that to Bertrandi is due the honour of having first recognized the possibility of rupture of the tunica vaginalis in hydroceles. Pott also described a case where traumatic rupture had resulted in spontaneous cure, and it is evident from his writings that Hunter fully recognized that rupture might occur. But, except for the publication of occasional cases of this accident, there has been but little notice taken of it in England or Germany, most of the literature of the subject being derived from Switzerland and France. So far about fifty cases have been recorded, but many of the earlier records, particularly of those treated by conservative methods, are exceedingly scanty. Nevertheless, it would appear that the condition can hardly be as rare as might be supposed, and that not a few cases pass unnoticed, for the three herein described have come under the notice of the writer in less than four years at a single hospital.

CLINICAL HISTORY.

A case of rupture occurring in an acute hydrocele has been described by Saint-Martin; but, with this exception, in practically every recorded case the hydrocele had existed for some months, or more often for several years. The history of repeated tapplings given in the first two

of my cases is by no means the rule, and I have only found it mentioned in three others, all of which, strange to say, were recorded by English observers. As a rule, there appear to be no premonitory symptoms, nor is there at all frequently rapid enlargement of the hydrocele during the few weeks preceding rupture, as Reverdin thought. In one or two cases the rupture has taken place during sleep, or has passed unnoticed by the patient, as in my second case; but usually it has been accompanied by a feeling of something tearing or giving way. Pain is a much more variable symptom, for, while usually severe and described as cutting or stabbing, it is occasionally entirely absent at first. The pain, when present, lasts a variable time, and is but rarely accompanied by faintness or any general symptoms; indeed, in not a few cases the patient has gone on with his work a few minutes after the occurrence of the rupture, and only sought medical advice after some hours on account of continuous or increasing discomfort. The immediate effect of the rupture, noticed by the patient, is usually that the swelling of his scrotum has become larger and softer, and an examination during the first twelve hours generally reveals a more or less uniform œdema, absolutely confined to the affected side of the scrotum, which appears to have been translucent in most of the cases in which this point was investigated. The position of the testicle can generally be made out by palpation at this stage. After the first few hours the œdema slowly spreads to the penis, especially the prepuce, the opposite side of the scrotum, and even at times to the lower part of the anterior abdominal wall and perineum. More or less discoloration of the scrotal tissues from effused blood is generally to be seen within twelve or twenty-four hours of the rupture, and where a vessel of any size has been torn a subserous or subcutaneous hæmatoma will be produced. The scrotum is usually very tender when touched, and there is sometimes a recrudescence of the pain, which lasts two or three days, and may be so severe as to compel the patient to keep his bed. Bailly regards the persistence or recurrence of pain as a sure sign of the formation of a hæmatocele, and the consequent necessity of operative interference.

If untreated, the œdema generally begins to subside after a few days, and has usually entirely disappeared in a fortnight or three weeks; while the hydrocele, which has either become much smaller or has disappeared altogether, in most cases slowly refills. It is probable that cases, such as those described by Velpeau, in which the patient goes to bed with a well-marked hydrocele and wakes up next morning without any scrotal swelling at all, are to be explained by rupture without

hæmorrhage and rapid absorption of the effused fluid. In a few cases after rupture a soft area has been noted in some part of the scrotum, in the base of which an opening admitting one or two fingers could be felt through the skin. The absorption of the fluid is usually accompanied by slight fever and general malaise, and occasionally a feeling of sickness. In a few cases polyuria and even diarrhoea have been noted. Complications are extremely rare, but cases of gangrene and even fatal septicæmia have been reported.

PATHOLOGY.

In considering the pathology of this affection one has first of all to inquire *why* the tunica vaginalis ruptures. In most of the recorded cases the traumatism producing the rupture, if present at all, has been of a quite insignificant character, while we know that the normal tunica vaginalis is a membrane of some strength and capable of resisting considerable fluid pressure. This is shown by the experiments of Saint-Martin and Delore. The former observer, by injecting fluid under pressure into the tunica vaginalis of post-mortem subjects, found that one atmosphere was the least pressure under which he ever obtained rupture, while in seven cases out of fourteen he failed to obtain rupture at all. In each of the seven cases in which rupture occurred it was situated in the upper part of the tunica vaginalis. Delore, however, found the rupture almost as frequent below as above. When a hydrocele has formed the tunica vaginalis has still considerable strength, and Maucclair and Vinsonneau state that they have tried to produce rupture by pressure on the hydroceles of patients under chloroform, immediately before performing radical cure, but have failed. It seems clear, therefore, that, except under the influence of relatively severe traumatism, to undergo rupture the tunica vaginalis must be in some way changed. In what exactly this change consists there is some difference of opinion; but before mentioning the two principal views on the subject, it will be convenient to briefly allude to the anatomy of the part.

As is well known, the parietal portion of the tunica vaginalis is covered by several layers of tissue. Beneath the skin is the dartos, a thin, reddish membrane of unstriated muscular and elastic fibres, which, according to Barrois, consists of two separate layers, the outer surrounding both testicles, the inner and stronger forming a separate investment for each. In the middle line there exists a potential space, bounded on each side by the deep layer of dartos, and into this space

pathological effusions occasionally take place. Next comes a thin layer of loose cellular tissue called by the French the fascia of Cooper. On injecting fluid into the potential space formed by this loose tissue, it is found to be completely closed on the side of the thigh, but to communicate freely with the subcutaneous tissue of the perineum, penis, and anterior abdominal wall. Here pathological effusions collect much more frequently. Beneath this again is a strong fibro-muscular tunic consisting of three layers, more or less easily separable. It may be regarded as a fibro-muscular bag enclosing the testicle and cord, open only at the level of the internal abdominal ring. The lower portion enclosing the testis is by far the stronger. Separating the tunica vaginalis from this fibro-muscular coat is a layer of subperitoneal cellular tissue, in which blood vessels, mainly branches of the funicular artery, ramify. This tissue is much better developed above than below; indeed, in its lower fourth the tunica vaginalis becomes firmly united to the fibro-muscular layer. Pathological effusions are frequent in this situation. According to Béraud, the tunica vaginalis often presents finger-like diverticula, one or two centimetres long, which end blindly sometimes after piercing the fibro-muscular tunic. While they may exist in any part of the tunica vaginalis, they are said to be most common above. Although their existence is not admitted by all anatomists, they are regarded by Saint-Martin as the essential cause of rupture in hydroceles. Burdet and Delore, on the contrary, think that the cause should rather be sought in the softening and degeneration of the tunica vaginalis and its coverings. In comparing these two theories, it will be well to inquire into the usual seat of rupture and the condition of the tunica vaginalis found at operation.

Saint-Martin, writing in 1883, stated that, according to his observations, the rupture most commonly took place in the antero-superior part of the tunica vaginalis. Reverdin, writing in the same year, regarded the anterior and central region as the most common seat. Since this time many cases have been recorded, and, as a large percentage of these have been subjected to operative interference, the exact locality of the rupture has been determined with much greater exactitude than formerly, and has been shown to have been situated at almost all points in the tunica vaginalis in different cases. From an examination of the records of a number of cases, it would seem that the rupture is slightly more frequent on the anterior aspect than on the posterior, a fact perhaps explained by the adhesion of the tunica vaginalis posteriorly to the scrotum noted several times. Some French authors seem to regard the

point of application of the traumatism in traumatic cases as important in determining the situation of the rupture, but it would appear contrary to the laws of fluid pressure to expect this. We must conclude that the tunica vaginalis ruptures at its weakest point, which may be anywhere, and by no means exclusively in the juxta-funicular portion, as Saint-Martin thought.

In the second place, what is the condition of the tunica vaginalis? In nearly every case in which the tunica vaginalis has been carefully examined it has been found diseased. The membrane itself is usually thickened and vascular, and may present on its inner aspect flattened plaques, of a yellowish colour, resembling those of atheroma, and sometimes calcified, or even ossified. In the substance of the membrane may be areas of fatty degeneration, and occasionally false membranes are found on its inner surface. Histological examination of sections of the thickened tunica vaginalis, after rupture, have shown it to be composed of fibrous connective tissue, so thick and dense as to be completely deprived of cellular elements. Reverdin, who describes two cases of comparatively short duration before rupture, notes that even in these there were lesions in the tunica vaginalis which no one would have expected, and the conditions found at operation, in my third case, are entirely in accordance with this observation. No doubt, where the applied traumatism is sufficiently severe, rupture may occasionally occur in tunicae vaginales that are but little changed, and it seems probable that the cases recorded by Stitelet were mainly of this nature. Even in these cases, however, a "washed-out" appearance of the tunica vaginalis is described. Thickening of the fibro-muscular covering has also been noted in a few cases, but whatever changes may take place in the tunica vaginalis, and the subjacent fibrous tunic, the two membranes are always easily separable from one another. It would appear, therefore, that the tunica vaginalis gives way because it is diseased, and that the rupture may occur at any point on its surface.

We must next inquire what it is that gives way when a rupture occurs. Is it the tunica vaginalis only, or does the fibrous layer rupture at the same time also? Reverdin was of opinion that both coats generally gave way, but Burdet considered that it was the tunica vaginalis only which usually ruptured. Delore's experiments, in which fluid under pressure was injected into the vaginal sac, seem rather to favour the latter view, for in one case only out of nine was the fibrous layer ruptured; but little reliance can be placed on the results of these experiments, since healthy tissues only were involved in them.

Certainly, in some of the more recent and carefully described cases it is the tunica vaginalis only which has given way, but there can be no doubt that both varieties occur; so that the effusion of the serous fluid and blood may take place, either into the subserous tissue or into the loose cellular tissue immediately beneath the dartos, or into both. Poncet, of Lyons, thinks it is possible to distinguish clinically between these two varieties. He says that a rapidly spreading œdema, with considerable ecchymosis, extending on to the abdominal wall, indicates a rupture of the fibrous tunic, while a limited swelling, accompanied by slight ecchymosis, will be in favour of a rupture of the tunica vaginalis only. This will be more certain if the effusion is prolonged along the cord and forms a tumour of some size. In ten days or a fortnight after the rupture the greater part of the effused fluid will have been absorbed, and if there has been much bleeding it is then quite easy to distinguish a vaginal from an extra-vaginal hæmatocele, for the former surrounds and obscures the testicle, while the latter forms a swelling to one or other side of it. When the scrotal skin is adherent to the tumour, it is strong evidence that the hæmatocele is situated external to the fibro-muscular coat. The hæmatocele thus formed may occasionally be absorbed, but it is much more usual for the scrotal tissues to become thickened around and for the swelling to persist indefinitely. Sometimes, in cases of repeated rupture without much hæmorrhage, an intra-parietal pocket is formed, which remains connected with the cavity of the hydrocele, or may be gradually separated from it.

The rupture itself is usually slit-like, with sharp-cut or jagged edges. As in the cases here described, it is often longer than one would have anticipated. The fluid found, on operation, in the sac of the hydrocele is usually blood-stained, and contains a variable amount of blood-clot. The pouches resulting from the rupture are also filled with clot. The older French observers attributed the hæmorrhage to rupture of vessels in the cellular tissue, resulting from excessive traction as this tissue became distended with effused hydrocele fluid. This may be true in part, but there can be no doubt that blood is also derived from the vessels divided by the rupture of the tunica vaginalis, or fibrous coat, the latter being often very vascular. Walther describes a case in which, on the evacuation of the clots, a profuse discharge of blood poured out from an artery in the torn edge of the tunica vaginalis. When the case is left to nature the tear in the tunica vaginalis, as a rule, slowly heals by the formation of a fibrous scar, and the hydrocele refills.

CAUSES.

The exciting causes of rupture may be traumatism, as by a knock or blow which presses the distended tunica vaginalis against the resting pubes; or pressure, as when the hydrocele becomes squeezed between the thighs by the contraction of the adductor muscles. There is also considerable evidence to show that muscular effort such as occurs during a fall, a cough, defæcation, or a fit of anger may produce the same effect. While Kocher would deny the possibility of this, Le Dran maintains it strongly. He believes that either by direct action of the lower fibres of the internal oblique on the vessels of the cord, or by increased intra-abdominal pressure, the tension in the blood vessels of the tunica vaginalis is raised to such an extent that they give way, and the consequent increase of fluid in the tunica vaginalis is sufficient to determine its rupture. Baseil, on the other hand, believes the contraction of the cremaster to be the chief causative factor in these cases. But, besides these, so-called spontaneous cases undoubtedly occur, where rupture takes place without any apparent cause. In Velpeau's case a rupture occurred while the patient was asleep, and in a still more curious case described by Reverdin the patient felt a sharp pain, followed by all the symptoms of rupture, while seated quietly in a chair. The existence of spontaneous cases is of great interest, and points strongly to some degenerative condition of the wall of the sac as the essential cause of rupture. In a few cases of this nature a rapid increase in the size of swelling has been noticed before the rupture, but this is, however, rare. It is probable that muscular effort is the commonest exciting cause, and that spontaneous ruptures are the rarest; but it is necessary to remember that most of the recorded cases occurred in hospital patients, who, for the most part, work hard, and are therefore most subject to traumatism and muscular strain. If, as seems probable, the true cause of rupture is degeneration of the wall of the hydrocele, in the absence of traumatism this degeneration may advance so far that rupture occurs after very slight violence, or even spontaneously.

DIAGNOSIS.

There is usually but little difficulty in recognizing this condition if a reliable history is obtainable. It seemed obvious from the first in my three cases. Elephantiasis scroti and extravasation of urine are said to be the conditions for which it has been mistaken, and its resemblance

to the latter is certainly close at first sight. Where rupture of the tunica vaginalis occurs alone, and where there is but little scrotal effusion, the condition may closely resemble a hæmatocele; indeed, some think that not a few cases diagnosed as vaginal or parietal hæmatoceles arise in this manner. It is also necessary to remember that a vaginal hæmatocele may itself rupture, and, in the absence of a reliable history and of translucency after the accident, the two conditions will be very difficult to distinguish. When the rupture comes on acutely, with fever and sickness, a strangulated hernia may be closely simulated. But the freedom of the hernial rings will at once make the diagnosis clear. Erysipelas will also have to be considered in such cases, but we must recollect that erysipelas may occasionally occur as a complication of rupture.

TREATMENT.

The first recorded case of ruptured hydrocele was published in 1757, and for many years very little in the way of treatment was recommended. But palliative measures rarely result in a permanent cure, as is seen by the records of several cases: for example, a patient of Serre's, who repeatedly ruptured his own hydrocele by pressure, when it became uncomfortably large, thereby obtaining temporary relief. Only two or three cases of cure without operation have been recorded years after rupture, nor should we anticipate that a simple rupture of a diseased organ would make it healthy. Injection of coagulants is likewise uncertain. While it has sufficed to cure in some cases, it has been followed by suppuration and sloughing in others. Where antiseptic precautions can be taken, the best treatment in most cases is to cut down on the hydrocele, and perform a radical cure, clearing out all the blood-clot, and removing the greater part of the parietal portion of the tunica vaginalis. Where, as sometimes happens, firm adhesions exist posteriorly, binding the tunica vaginalis to the scrotum, it is probably best to follow Reverdin's advice, and leave this area untouched. In old men, too, where the testicle has ceased to be a functional organ, castration may be the simpler course. The only question is as to the best time to operate. When the œdema and infiltration are considerable, as in ruptures beneath the dartos, Poncet, of Lyon, recommends waiting a few days or a week, till most of the fluid is absorbed. He says the danger of suppuration, which is so liable to occur with large effusions of serum and blood, is thereby lessened. But where there is great pain from distension something must be done to relieve the patient, and

Bailey suggests that aseptic puncture of the hæmatocele is desirable as a temporary measure. With limited effusions there can be no reason to postpone operation.

In conclusion, I should like to thank Mr. Bland-Sutton for permission to publish the following cases, upon which, as well as those referred to in the following bibliography, the paper is based:—

Case I.—H. F., aged 52, a painter, had suffered from a hydrocele of the left tunica vaginalis for five years. During this time he had been accustomed to have it tapped about every six months, the last occasion being in May, 1905. As more than the usual time between the tapplings had elapsed, and as the hydrocele, though painless, gave trouble through its size and weight, he had determined on the morrow to seek relief. On March 9, 1906, at about 10.30 a.m., the man, who was out of work at the time, was walking down Charing Cross Road, and, seeing a sixpence, bent down quickly to pick it up. In doing this he is quite sure that he did not knock his hydrocele, but he thinks that possibly he may have pressed on it as he stooped down. He immediately experienced a sensation of something tearing or giving way, and felt a sharp pain in the left side of his scrotum. He felt rather giddy for a few minutes; but as soon as he recovered he felt for his hydrocele, which before had been smooth, firm, elongated from above down, and confined to the left side of his scrotum, and noticed that it had become quite soft. He walked about for a time, but as the pain, which had never left him since the accident, increased in severity, and as he began to feel generally ill, he came to the Middlesex Hospital at about a quarter to twelve. When seen in the out-patient room about 2.30 his condition was as follows: The left side of the scrotum was swollen to the size of a large orange. There was considerable superficial œdema, so that the skin appeared irregular on the surface, like the skin of an orange, and pitted readily. The œdema was entirely confined to the left side of the scrotum, and there was no discoloration or sign of effused blood anywhere. The whole scrotum was soft, and, although on account of the tenderness the outline of the left testis could not be made out, it was quite evident that the scrotum contained no tense swelling of any appreciable size. Over the left external abdominal ring was a pad of fat. The penis was quite normal. The next day slightly increased swelling of the left side of the scrotum was noticed, as well as some œdema of the right. There was also some ecchymosis in the skin, especially on the left side, and both œdema and discoloration of the penis, most marked at the preputial fold. Mr. Bland-Sutton operated the same afternoon. An incision about 3 in. long was made over the anterior aspect of the left side of the scrotum. The skin and dartos were œdematous, and contained a good deal of effused blood of a dark colour. When the tunica vaginalis was incised it was found to contain about an ounce of blood-stained fluid. On its posterior aspect, behind and outside the testes, was a vertical tear some 2 in. or $2\frac{1}{2}$ in. long and almost straight. The edges of this tear appeared clean-cut and not jagged,

174 Hastings: *Rupture of Tunica Vaginalis in Hydroceles*

and beyond it was a cavity in the cellular tissue of the scrotum, containing some amount of clotted blood. This was cleared away and a radical cure performed. The greater part of the parietal layer of the tunica vaginalis was removed and the testicle invaginated into the remainder, which was secured around it by two stitches. A drainage-tube was inserted, and, on account of the oedema, two stitches only were used to unite the skin. The oedema rapidly became less, and on the fourth day after operation had so far decreased that five extra superficial stitches were added. The wound healed slowly without suppuration, and the patient was discharged on April 4. Unfortunately, no microscopic examination of the tunica vaginalis was made in this case.¹

Case II.—The second case is that of a journeyman baker, aged 43. Except that he had had a left-side hydrocele for five years, there was nothing noteworthy in his previous history. The man had a sallow, unhealthy appearance, the result of his employment, and his arteries were rather thickened. He stated that he had never had syphilis, and that he was a moderate drinker, but occasionally, as he expressed it, "went on the spree." He had had his hydrocele tapped in June, 1906, when 22 oz. of fluid were removed, and again in March, 1907, when 20 oz. were drawn off. The hydrocele had again filled, and extended as high as the internal abdominal ring, the swelling being most marked above. It was beginning to cause him inconvenience by its size, and was slightly tender, so that he had made up his mind to come to the hospital in a few days' time to have it tapped again. He has no recollection of ever knocking the hydrocele, but on April 11, 1908, he noticed that the swelling had become softer and more tender than usual. On April 12 this condition became more noticeable, and the chief swelling being in the lower part of the scrotum instead of above as previously. To quote the man's own words, his hydrocele "had dropped all flabby like a busted football." There was also discoloration of the skin on both sides of the scrotum. On April 13 the pain became so severe that the man was unable to continue at his work, and came to the Middlesex Hospital. The scrotum was then found to be oedematous, especially on the left side. The right testicle could be felt, but the left could not be made out, although testicular sensation was obtained on both sides. Neither fluctuation nor translucency was present. The skin of the scrotum was discoloured on both sides, but especially on the left. The next day (April 14) all oedema on the right side of the scrotum had disappeared. The left testis could not be made out, but fluctuation was obtained, and the fluid could be displaced from the lower part of the scrotum to the external abdominal ring. On April 23 the patient was admitted to the hospital. Practically all oedema and discoloration of the skin of the scrotum had disappeared. The hydrocele felt tenser than before. On April 29, through the kindness of Mr. Bland-Sutton, I performed a radical cure of the hydrocele. An incision 2 in. long was made over the upper part of the left side of the scrotum. A thick-walled hydrocele sac was opened.

¹ The above case has been previously published in the *Archives of the Middlesex Hospital*, 1906, viii, p. 35.

The contents of the sac were clear serous fluid, and the inner surface of the sac was smooth generally, but nodular where it covered the testis. The wall of the sac was everywhere thick, and consisted of a double membrane. To the inner side of this sac was a second nearly as large as the first. It was subcutaneous and lined by a thin serous membrane. It contained blood-stained fluid, and had ruptured posteriorly. The septum between the two sacs was thick and dense, but at one point it was much thinner and bulged toward the thin-walled sac. As much as possible of both sacs was dissected away, and a running stitch applied round the cut edge of the tunica vaginalis where it was reflected on to the testis, to limit hæmorrhage. The serous surface of the testis was then wiped with raw carbolic and the wound closed. A microscopic examination of the thickened tunica vaginalis showed it to consist of dense fibrous tissue with thick-walled arteries. The fibrous tissue was moderately cellular and contained elongated cells, with abundant protoplasm and a small round nucleus. It is quite clear that in the above case it was the thin-walled sac and not the tunica vaginalis that had ruptured. It would seem probable that this sac was secondary and due either to a previous rupture of the tunica vaginalis and the fibro-muscular coat, or to a leakage after tapping. It may be that the thin bulging area noted in the intervening wall between the two sacs represented a previous communication between them.

Case III.—On April 3, 1909, J. C., a shop assistant, aged 24, was admitted to the Middlesex Hospital for ruptured hydrocele. There is nothing special in his family or previous history. About a year before admission he noticed a swelling in the left side of his scrotum. It appeared spontaneously, and steadily increased in size. In about three months, though there was no pain, it became so inconveniently large that he consulted a doctor, who pronounced it a hydrocele, and tapped it. For two months all went well, but after this the hydrocele began to fill up again, and, after attaining the size of an orange, remained stationary. On April 3, while leaning over a billiard table, the young man knocked his scrotum. The blow was quite slight, but immediately he felt an acute pain, which, as he expresses it, "doubled him up." In a few minutes the pain became less acute, so that he was able to finish his game, but a dull aching followed. He examined his scrotum, and, finding it swollen and discoloured, came at once to the hospital and was admitted. On admission the scrotum was seen to be swollen to the size of a cocoanut, and discoloured by extravasated blood. The discoloration was general, though most marked below, but the swelling was almost confined to the left side. The scrotum was very tender. The swelling was tense, and fluctuation was obtained in it, but it was translucent. The testicle could not be felt. There was some œdema of the skin of the penis and prepuce, which were slightly discoloured. On April 5 Mr. Bland-Sutton decided to operate. A vertical incision 3 in. long was made downward from a point a little below the left external abdominal ring. Much blood-clot was found in the scrotal tissues. The tunica vaginalis was opened by deepening the incision, and found to contain blood-stained fluid and

clot. When this was removed a vertical tear some 2 in. long was seen extending through the parietal layer of the tunica vaginalis on the outer side. A portion of the thickened tunica vaginalis was removed, and the remainder everted and sutured behind the testis. A drainage-tube was inserted and the wound closed. The man made an uninterrupted recovery. On April 21 he left the hospital, the wound being completely healed. Microscopic examination of portions of the thickened tunica vaginalis removed at operation two days after the rupture showed it to consist of dense fibrous tissue. Between the fibres were a fair proportion of elongated fibroblasts and small round cells. The rather numerous blood vessels were in no way thickened, but were immediately surrounded by a large number of small round cells. It is interesting to note that the thickening of the tunica vaginalis was by no means uniform, being much more marked in some parts of the membrane than in others.

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DISCUSSION.

The PRESIDENT (Mr. Godlee) asked whether, in all the recorded cases, there was anything to point to disease of the testicle, such, for example, as syphilis, as hydroceles were frequently associated with some disease of that organ. He thought it must be a rare condition, as he had not come across a case in a somewhat long experience. And the only cases he could think of that might have been confused with it, which he had seen, were those which were formerly called inflammatory œdema of the scrotum, or erysipelas of the scrotum, which cases were comparatively common many years ago, but of which he now seldom saw an example.

Mr. SOMERVILLE HASTINGS, in reply to the President, said he had never found any record of any disease of the testicle in those cases (about forty in all) of which he had seen the reports, nor had syphilis been mentioned in any of them. There was no syphilitic history in any of his three cases, because he had inquired carefully about that point in all of them.

The Clinical Significance of Gastric Hypersecretion and its Connexion with Latent Disease of the Appendix.

By W. SOLTAU FENWICK, M.D.

THE disorder known as "hypersecretion" is distinguished from all other functional perversions of the stomach by the fact that a secretion of gastric juice occurs in the intervals as well as during the progress of digestion, and is therefore practically continuous. The symptoms of this disorder seem to have been quite familiar to English physicians during the early part of last century, who were wont to ascribe them to an "acid" or "irritative" condition of the stomach; but it was not until the publication of Reichmann's researches in 1882 that the association of these clinical phenomena with a continuous secretion of gastric juice became clearly recognized. But, although this writer proved conclusively the existence of what he termed "gastrosuccorrhœa," considerable doubts have been expressed as to its exact significance, while Schreiber and his followers have contended that, owing to the stimulating influence upon the gastric mucosa of dust, saliva and mucus, which are swallowed unconsciously during the night, a healthy fasting stomach always contains a certain amount of secretion in the early morning.

The experiments upon which these latter statements were based appear, however, to be open to criticism, since the stomach was evacuated by syphonage aided by pressure applied to the epigastrium and by voluntary efforts at straining on the part of the patient. Considering how difficult it is completely to empty the stomach after lavage, and how much saliva is usually swallowed during the process, to say nothing of the gastric irritation induced by the presence of the tube, it is hardly a matter for wonder that a conscientious attempt to investigate a fasting stomach with a tube and syphon usually succeeds in withdrawing a small quantity of fluid from the viscus. The employment of an evacuator, like that invented by Gentile, serves to eliminate many of these sources of error, since it permits the complete evacuation of the stomach within thirty seconds, during which brief time it is hardly conceivable that more than a few drops of secretion could be produced. Experiments conducted on these lines invariably show that when a healthy stomach is carefully washed out and emptied overnight and again aspirated in the early morning, no food or drink having been taken in the interval, it is never found to contain more than a few cubic centimetres of a neutral or faintly acid fluid. This conclusion tallies so closely with the results obtained by Riegel and other modern investigators who have employed aspiration instead of syphonage that the existence of more than 20 c.c. of gastric juice in the fasting stomach must be considered indicative of disease. When, however, a typical case of hypersecretion is examined in the same manner a very different picture is presented. Thus, if the stomach be washed out and emptied overnight, and no food or drink be taken in the interval, aspiration on the following morning will show the organ to contain from 50 c.c. to 300 c.c. of an opalescent or bile-stained fluid which gives all the reactions of an active gastric juice with a total acidity varying from forty to seventy. The mere withdrawal of its contents appears to stimulate the fasting organ to fresh secretory exertions, for if aspiration be repeated hour by hour a similar quantity may be drawn off on each occasion. It is obvious, therefore, that in certain cases the stomach continues to secrete an active juice without the normal stimulus supplied by the ingestion of food. In the light of this fact the symptoms that usually accompany hypersecretion are easily explained. In mild instances, where the stomach contains only a small quantity of fluid in the early morning, very little discomfort is experienced before breakfast, but in the more severe types of the complaint, fullness, distension, nausea, giddiness, and perhaps acid eructations, ensue immediately the patient rises from bed and may even awaken him before the usual time.

The special symptoms of hypersecretion do not usually display themselves until the lapse of two or three hours after breakfast, when, the proteid constituents of the meal having undergone solution and partial elimination into the duodenum, the percentage of free hydrochloric acid attains the point at which it is able to excite extreme irritation of the gastric mucosa and spasm of the pyloric sphincter. Consequently, between eleven and twelve o'clock, the exact time varying with the size and nature of the meal, the stomach becomes inflated with gas and pain makes itself felt in the epigastrium or right hypochondrium. If recourse be had to some form of proteid food these various symptoms usually abate for a time owing to the chemical combination of the free mineral acid with the nitrogenous base, while a draught of water may produce similar relief by effecting dilution of the hyperacid contents of the organ. It is for this reason that so many sufferers from hypersecretion insist that their symptoms are not dyspeptic but arise from hunger, and, if pain exists, term it a "hunger pain." In like manner the midday meal is followed by a recrudescence of the symptoms between three and five o'clock in the afternoon, which are always intensified by a cup of tea and may not subside until after dinner. But the most characteristic attack is undoubtedly that which occurs between one and two o'clock in the morning, when, after some disturbed sleep, the patient will awaken to the fact that he has a pain in the upper part of the abdomen, which is followed by flatulence and acidity, and, perhaps, by vomiting. In these circumstances the ejecta are copious, liquid, bile-stained, extremely acid, and afford the usual reactions of gastric juice containing an excess of free hydrochloric acid. In addition to these general symptoms that are common to all forms of hypersecretion, three local results of the disorder deserve notice, as they help to explain some of its aberrant features.

In the first place, the constant excess of free hydrochloric acid sooner or later gives rise to a spasmodic closure of the pylorus, which makes itself evident not merely by pain but by an intermittent obstruction to the passage of chyme into the intestine. This condition, which may easily be demonstrated, can alone explain the constant presence of undigested food in the fasting organ in cases where operation proves the non-existence of organic stricture.

Secondly, the long-continued immersion of the delicate lining membrane of the stomach in a fluid that contains from two to five times the normal percentage of free hydrochloric acid excites a violent form of gastritis, which is evidenced at operation by an intense congestion of

the mucosa and numerous interstitial hæmorrhages and hæmorrhagic erosions. The last named are apt to ooze blood, and, when exceptionally numerous, hæmatemesis or melæna is usually observed. I have no hesitation in saying that in the vast majority of cases where bleeding points are detected in the gastric mucous membrane or moderate hæmorrhage occurs during life without obvious ulceration being found after death, the condition is due to chronic hypersecretion accompanied by superficial losses of tissue too minute to be detected by the naked eye. In one case of this kind which I had an opportunity of examining with von Recklinghausen we were able to demonstrate by artificial injection the source of the fatal hæmorrhage, which had escaped his most careful scrutiny at the autopsy. Although these erosions usually heal, they may, if numerous and closely set, ultimately coalesce and form superficial ulcers of considerable size, which, however, may not be recognizable on digital examination of the exterior of the stomach. I have seen an ulcer of this description, nearly 2 in. in diameter and extending as deeply as the muscular coat, which could not be detected until the stomach had been opened.

Thirdly, the amount of gastric secretion as well as its degree of acidity varies not only in different cases, but also at different stages of the same case. In addition to the well-marked remissions which characterize all forms of the complaint, the course of chronic hypersecretion is apt to be interrupted from time to time by an acute phase of the disorder, during which vomiting of a hyperacid gastric juice mixed with bile or altered blood is almost incessant, much pain is experienced in the region of the stomach, and dangerous exhaustion ensues. These intercurrent attacks are usually described as a distinct variety of the complaint under the terms "intermittent" or "periodic" hypersecretion, but inasmuch as they always co-exist with the chronic form and are associated with the same organic lesions this differentiation appears to be superfluous.

With regard to the frequency of hypersecretion as a cause of dyspepsia, I found that among 500 consecutive cases of chronic indigestion examined in hospital, 67, or about 13 per cent., were examples of this disorder, while a similar number of cases observed in private practice afforded 170 instances, or a percentage of 34. Although it is well known that chronic hypersecretion commonly accompanies chronic ulcer of the stomach, it is still the custom to regard it as a primary disorder of nervous origin and to attribute it to the same conditions that are supposed to excite simple hyperacidity. Thus, all writers lay much stress upon over-indulgence in

rich and highly spiced foods, the abuse of alcohol or tobacco, inefficient mastication, and long-continued mental strain or excitement; but since these several conditions are also held to be responsible in great measure for hyperacidity and gastritis, they cannot possibly be regarded as the sole exciting causes of hypersecretion. Until the year 1907 I had convinced myself from post-mortem evidence, as well as from the more limited data afforded by operation, that 88 per cent. of all cases of chronic hypersecretion were associated with a demonstrable lesion of the digestive organs, while in the remaining 12 per cent. no disease that appeared to have any immediate connexion with the stomach could invariably be detected. I was, however, well acquainted with a peculiar type of hypersecretion in which death frequently occurred from appendicitis, and was in the habit of warning the subjects of this complaint of their special liability to inflammation of the appendix; but it was not until an opportunity occurred in that year of discussing the subject with Dr. W. J. Mayo, of Rochester, U.S.A., that the cause of this appendicitis, and also an explanation of the 12 per cent. of cases hitherto unexplained, at once became apparent. That distinguished surgeon informed me that he had often discovered latent disease of the appendix in persons who appeared to require gastro-jejunostomy, and that the removal of the appendix was followed by subsidence of the gastric symptoms, provided that the alimentary tract was otherwise healthy. Furthermore, that several of his earlier cases of gastro-jejunostomy which he had not materially benefited by the operation had subsequently been found to possess disease of the appendix, and that when this had been removed a cure had ensued. With these facts in mind the various surgeons who have operated for me on cases of chronic hypersecretion during the last two years have examined the appendix as well as the other important abdominal viscera, and the results obtained in 112 consecutive cases are as follows:—

	Cases
Chronic ulcer of the stomach existed alone in	13
Chronic duodenal ulcer existed alone in	46
Gall-stones existed alone in	12
Disease of the appendix existed alone in	22
Gastric and duodenal ulcers co-existed in	3
Duodenal ulcer and gall-stones co-existed in	3
Gastric ulcer and diseased appendix co-existed in	5
Duodenal ulcer and diseased appendix co-existed in	4
Cancer of the pylorus existed alone in	4
Total	112

Out of the thirteen examples of gastric ulcer, the disease occupied the cardiac portion of the viscus in four, the central zone in three, and the pyloric third in six; and it is interesting to observe that one of the most severe clinical examples of hypersecretion was associated with an ulcer close to the cardiac orifice. When gall-stones constituted the sole indication of disease, the calculus was often single and completely filled the gall-bladder, and only in one case was there a history of biliary colic. Hæmatemesis or melæna was noted in only three out of the forty-six examples of duodenal ulcer, notwithstanding the fact that the average duration of the symptoms of hypersecretion prior to the operation was nine years, while in several instances they had existed more than seventeen years. In only seven of the twenty-two cases of diseased appendix was there a history suggestive of previous inflammation of the organ, although at operation it was invariably found to be either extensively ulcerated, thickened, adherent, dilated, or occupied by a calculus. In each of the four examples of cancer of the pylorus the growth was of the cylinder-cell variety, which developed with extreme rapidity and caused death within seven months. There was no history of former simple ulcer in any case.

The dependence of hypersecretion upon an organic lesion of the digestive organs appears to be further corroborated by the results of treatment. Thus in almost every case where gastro-jejunostomy was performed for gastric or duodenal ulcer, while the gall-bladder and appendix were proved to be healthy, the symptoms of disordered digestion gradually disappeared; and in those which were carefully examined subsequently by Paterson, the hypersecretion was found to have ceased. In like manner the removal of gall-stones, when other parts of the digestive tract were healthy, was followed by subsidence of the secretory disorder, while appendicectomy, after a preliminary increase in the amount of hydrochloric acid, was usually, though not invariably, attended by similar results.

A careful comparison of the clinical and chemical features of these various cases, with the lesions found at operation, appears to indicate that each division possesses certain general characters which are sufficiently distinctive to permit of a correct diagnosis being made in the majority of instances.

(1) Hypersecretion due to disease of the appendix, or "appendicular hypersecretion" as it may be termed, is the mildest form of the complaint. As a rule, it displays an intermittent character for several years and each fresh attack is ascribed by the patient either to a chill, mental

anxiety, or to some indiscretion of diet. For a considerable time after it has attained the chronic stage, the typical symptoms of a moderate hypersecretion continue to manifest themselves; but with the progress of time, the paroxysmal pain is gradually replaced by discomfort and distension, which persist during the entire day. So far as my limited experience goes, the symptoms vary to a great extent according to the nature of the appendicular lesion, and the concomitant state of the gastric secretion. Thus, when the appendix is in a state of active irritation such as ensues from ulceration of its mucous membrane or the presence of a calculus, pain is always a noticeable feature, and a gastric analysis shows both hyperacidity and moderate food retention. On the other hand, if the appendix is merely thickened or adherent, owing to a former attack of acute inflammation, the symptoms of the gastric disorder are identical with those of chronic inflammation of the stomach, and subacidity, with an absence of free hydrochloric acid, is the rule. In every instance of this latter kind that has come under my notice the patient was thin, anæmic, and despondent, and was obviously suffering from general neurasthenia. Although the complaint had persisted for several years, no form of diet or medicinal treatment had been discovered which produced any permanent benefit, and it is worthy of remark that the patients often stated that bicarbonate of sodium and other alkalies gave rise to pain, or otherwise disagreed with them. Cases of this description endure a miserable existence and usually manage to exhaust both the sympathy of their friends and the patience of their medical attendants by their constant and apparently imaginary complaints. Nevertheless, when carefully examined, several indications may be forthcoming which suggest the nature of the malady. In every instance the stomach is moderately dilated and dislocated downwards in the abdomen, and there is an increased sensitiveness to pressure over the entire organ. Sometimes palpation of the epigastrium gives rise to a pain which radiates towards the right iliac fossa, or pressure over the latter region produces pain above the umbilicus; but these phenomena, though very suggestive when present, only occur in a limited proportion of the cases. As a rule, no history can be obtained indicative of former appendicitis, but occasionally the statement will be volunteered that the indigestion started with inflammation of the bowels, peritonitis, or intestinal stoppage. Aspiration in the early morning usually shows the stomach to contain 10 c.c. to 50 c.c. of thick mucus, or of a weak gastric juice mixed with mucus, but in some instances the organ is almost empty. After a test

breakfast the material evacuated exceeds the normal in quantity, is difficult to filter on account of mucus, is practically devoid of free hydrochloric acid, and rarely possesses a total acidity of more than 50. "Nervous dyspepsia" is the term most commonly applied to this form of the complaint, although in true gastric neurasthenia both secretion and motility are normal; while other diagnoses refer the symptoms to biliousness, gastropnoia, hypochondriasis, or hysteria. Sooner or later, however, these unfortunate individuals develop an acute, and usually suppurative, appendicitis, and it was the frequency of this complication that first drew my attention to the subject.

(2) *Biliary hypersecretion* is more frequent in women than men, and rarely develops before thirty years of age. It is most severe when the gall-bladder is occupied by a single large stone, and for this reason, probably, is seldom preceded by colic or jaundice. The disorder displays the same tendency to intermissions as the other varieties, but each individual attack often lasts for two or three months. Pain during the later stages of gastric digestion is invariably present, and is usually referred to the right hypochondrium, whence it may radiate to the back of the chest. Vomiting may or may not occur, but acid regurgitations constitute one of the most prominent features of the case. The so-called "hunger pain" is often very pronounced. In the early morning the stomach contains a considerable quantity of bile-stained gastric juice, which possesses a comparatively high total acidity and affords the usual reactions of free hydrochloric acid. After a test-breakfast the total acidity varies from 70 to 110, and the percentage of free hydrochloric acid is greatly increased (0.04 to 0.1). The fact that biliary hypersecretion is often followed by ulcer of the stomach or duodenum renders its differential diagnosis from these latter complaints a matter of considerable difficulty. The chief points in favour of primary biliary calculus are its greater frequency in women, the persistency and severity of acid regurgitations, and constant tenderness over the region of the gall-bladder.

(3) Hypersecretion due to an ulcer in the vicinity of the pylorus, whether on its gastric or duodenal side, is accompanied by the various symptoms of the disorder in their most characteristic form. Whatever may be the expression employed to define the digestive discomfort that occurs in other varieties, the subjects of this form of the complaint state most emphatically that they suffer intense pain, which usually develops about two hours after a meal, and persists until the next, unless relieved by vomiting. In all cases the nocturnal attacks are the most severe and are often accompanied by vomiting of the characteristic bile-stained gastric

juice. Sooner or later emesis occurs at other times of the days, emaciation sets in, and the case is exposed to such dangerous complications as hæmorrhage, tetany, and acute hypersecretion. Dilatation of the stomach is always in evidence, and if the ulcer has produced stenosis of the pylorus or duodenum, peristaltic movements of the organ may be visible on inspection of the abdomen. It is in these cases that the stomach is found to contain a large amount of a hyperacid gastric juice, mixed with undigested food, in the early morning, and its contents, after a test-breakfast, to exhibit a great increase of free hydrochloric acid.

CONCLUSIONS.

(1) Chronic hypersecretion of the gastric juice is not a primary functional disorder of the stomach, but is invariably the result, possibly a reflex one, of organic disease of the stomach itself, or of some other organ of the body. In the great majority of cases it is found to be associated either with ulcer of the stomach or duodenum, gall-stones, or disease of the appendix; but it is probable that future investigations will show that pancreatic calculus, tubercle of the cæcum, and new growths of the appendix are also capable of producing it.

(2) A continuous flow of a hyperacid gastric juice, whatever be its cause, always excites severe inflammation of the stomach, and is very liable to be followed by ulceration of that organ or the duodenum. This secondary ulceration is at first quite superficial in character, and may not be demonstrable on examination of the exterior of these viscera. Minute hæmorrhagic erosions, capable of producing hæmatemesis and mælena, are often associated with, and caused by, this abnormality of secretion.

(3) There is an important connexion between the condition of the appendix and the state of the gastric secretion. When active irritation of the former exists, such as ensues from ulceration of its mucosa or the presence of a calculus, the gastric juice is usually increased both in quantity and acidity, and the patient exhibits the usual phenomena of hypersecretion. On the other hand, thickening, torsion, cystic dilatation, or adhesion of the appendix, which result from an attack of acute inflammation, are apt to be followed after a time by a peculiar type of chronic gastritis, attended by persistent flatulence, nausea and anorexia, which the usual methods of treatment fail to allay. In this condition the contents of the stomach exhibit an excess of mucus, and are usually devoid of free hydrochloric acid.

(4) With the exception of severe hæmorrhage, all the symptoms of chronic duodenal ulcer are due to the hypersecretion which accompanies it. The pain that ensues two or three hours after the ingestion of food is probably caused by contact of the sore with the hyperacid contents of the bowel. That the presence of chyme, or undigested food, is not essential to the production of pain is evidenced by the characteristic seizures that occur during the night, when the stomach is devoid of food, or after oral feeding has been suspended for several days. The constant immersion of the ulcer in a relatively strong solution of free hydrochloric acid is one of the chief obstacles to its healing. The so-called "hunger pain" is met with in all the clinical forms of hypersecretion, but since its severity varies with the degree of acidity, it is usually most pronounced in cases of ulcer of the stomach or duodenum.

(5) Contrary to the general belief, a soft carcinoma of the pylorus is sometimes attended by severe hypersecretion. In such cases the morbid growth often develops with great rapidity, and violent hæmorrhages are apt to occur, possibly owing to digestion of the growth. In many instances the history affords no indication of previous simple ulcer.

Appendicular Gastralgia, or the Appendix as a Cause of Gastric Symptoms.¹

By HERBERT J. PATERSON, F.R.C.S.

DURING a visit to America in the spring of 1907 I spent two weeks at the famous Mayo clinic, and while there saw Dr. W. J. Mayo operate on two patients who were supposed to be suffering from gastric or duodenal ulcer. The history and symptoms were clear, but in neither patient was any gastric lesion discovered at the operation. In one the appendix was chronically inflamed, and in the other it contained a concretion. In both cases Dr. Mayo performed appendicectomy, and stated his belief that the appendix was the cause of the gastric symptoms, which he attributed to reflex spasm of the pylorus produced by the condition of the appendix. I must confess that at the time I was somewhat sceptical—the extent of the appendicular disease did not seem to me to be adequate to account for the severity of the gastric symptoms—but I have since from my own experience been able to confirm the soundness of Dr. Mayo's judgment, although, as I shall point out later, I am doubtful as to how far pyloric spasm accounts for the symptoms observed.

That there are cases in which the disease of the appendix mimics gastric disease, in some cases with very remarkable fidelity, does not admit of doubt. This does not appear to have obtained general recognition in this country,² although probably the condition has been recognized by some. Abroad it has been recognized for some years. Ten years ago Ewald [2] referred to such cases under the name "Appendicitis larvata," and Senator used the term "Atypische Appendicitis" for them. In America the rôle of the appendix in the production of gastric disorder has been recognized not only in the work of the Mayos, but in the writings of Murphy, Ochsner, Deaver, and others. Personally I have operated on twenty-four such cases, and I suggest that the term "appendicular gastralgia" is a convenient and descriptive

¹ Towards the expenses of the researches referred to in this paper, a grant was made by the Science Committee of the British Medical Association.

² Since this paper has been written and accepted for reading Mr. Moynihan has published a paper (*Brit. Med. Journ.*, 1910, i, p. 241) on the same subject, which has led to an interesting discussion in the columns of that journal.

one to apply to them. In the majority of them operation was undertaken for a supposed gastric or duodenal ulcer, but later experience has led me to believe that in many instances it is possible to make a correct diagnosis, and this was the case in seven of my last eight cases. Six of these were seen with my colleague, Dr. Soltau Fenwick, and in all but one we made a correct diagnosis as to the appendicular origin of the gastric symptoms. The following selection of cases is illustrative of the various symptoms and different conditions found :—

(1) Female, aged 29. At the age of 16 had jaundice. Since she was aged 20 she has suffered from attacks of gastritis. One child in July, 1902; laid up with "peritonitis" for six weeks after confinement. Since June, 1907, has suffered continuously with indigestion, with occasional attacks of very severe pain, coming on about one hour after food. Pain referred to a spot just to right of umbilicus. Has felt sick at times, but has had no vomiting. Occasionally she has a bitter taste in her mouth. Has been on an almost exclusive milk diet. For the last month has had to remain in bed owing to sickness and feeling of being completely worn out. Patient is anæmic, with a very sallow complexion, and has evidently lost flesh. No free HCl in gastric contents, and marked excess of volatile acids. Patient was too ill for operation when first seen, but improved a little on a purely milk diet, and the administration of saline injections by the rectum. Operation, December 1907: Stomach and duodenum dilated; no sign of ulcer anywhere; appendix bound down behind the cæcum with dense adhesions, 5 in. long, and bent on itself at an acute angle, so that two divisions were lying almost parallel. Appendicectomy: On opening the appendix a concretion was found in distal half; the mucous membrane of proximal half was ulcerated. Uninterrupted recovery. Last report, January 1910: Patient has remained perfectly well since the operation, and has been able to follow continuously her usual employment.

(2) Female, aged 32, sent to me by Dr. F. J. Barker. For six years she has always suffered from pain after food. During this period she has had at intervals attacks of pain, sometimes severe, across the upper part of the abdomen. At first she did not have to lie up during these attacks, but during the last five months she has had three attacks, during which she had had to lie up. These last three attacks have been accompanied by vomiting. The last attack commenced with pain over the pubes. Operation, December 4, 1907: Long contractile appendix, no adhesions, but containing a largish concretion; much congestion of mucosa. Microscope: Much infiltration of all appendicular coats. Patient seen February, 1910: She has remained perfectly well since the operation, and has been able to do her work uninterruptedly.

(3) Male, aged 32. Quite well until May, 1907, then sudden pain in left side of abdomen, accompanied by vomiting. In bed for three weeks. Tenderness about umbilicus persisted for some weeks. Ever since this attack has suffered from severe pain, coming on two to three hours after food. Has had

to go to bed on several occasions owing to increased severity of this pain. Has lost several pounds in weight. Tenderness in epigastrium, stomach dilated. Tenderness in both iliac fossæ, more marked in left. January, 1908, operation: Appendix much thickened and bulbous; internally, mucous membrane much congested and ulcerated in one place. December, 1909: Patient has remained quite well since operation, and has had no recurrence of pain.

(4) Female, aged 27. Quite well until three years ago. Since that time she has been subject to occasional attacks of pain just above the umbilicus, accompanied by flatulence and vomiting. These attacks were thought to be due to an inguinal hernia, and she was operated on for this and a radical cure performed in April, 1908. She was no better after the operation, and the attacks have continued. The last attack was in April, 1909, when she was laid up for seven weeks. Since this attack she has suffered from occasional pain in right iliac fossa. Tenderness on pressure in epigastrium and also in right iliac region. Operation, June 7, 1909: Appendix adherent to omentum. Appendicectomy: Appendix $3\frac{1}{2}$ in. long, mucous membrane much congested and petechial over greater extent. Last report, February, 1910: Patient reports herself as quite well ever since operation, except for an occasional throbbing pain in the wound.

(10) Female, aged 29. Three years ago had an attack of severe pain in upper part of abdomen. Since then has been subject to severe attacks of pain after food. These attacks have recurred at intervals of about a month, and have been especially severe when occurring during a menstrual period. Occasionally she has vomited. During the intervals between the attacks she has invariably suffered from bad indigestion. The pain is referred to the umbilicus and upper abdomen, and comes on directly after taking food. During the last attack the pain extended downwards towards the right side. Gastric analysis: Ten hours after lavage and milk stomach contained 50 c.c. very thick brownish fluid; total acidity, 22; no free HCl. Test meal, 70 c.c.—Very thick and tenacious gastric contents, containing much mucus; total acidity, 30; volatile acids, 14 per cent.; total chlorides, 0.179; free HCl, *nil*; protein HCl, 0.111; mineral HCl, 0.068; digestion, none. Operation, January 1909: Appendix $6\frac{1}{2}$ in. long, much thickened, and adherent to right ovary, which was cystic. Appendicectomy. Symptoms gradually disappeared, and two months later patient was quite well and had had no further pain. Later report, February 20, 1910: Patient says that she has quite regained her usual health and strength, and has had no more indigestion.

(12) Female, aged 21. Patient's illness began two years ago with pain in the stomach immediately after taking food. Since that time she has always had pain immediately after taking food. She has had no vomiting, but has had sour eructations. The pain is referred to the epigastrium. There is slight tenderness to right of umbilicus one-third of way to anterior superior spine. No treatment has had any effect on the pain. Operation, May, 1909: Appendix long, some adhesions fixing it to cæcum, bulbous at distal end; internally, contained faecal material, mucosa congested. Microscope: Lymphoid tissue is

abundant, and there is much small-celled infiltration of the mucous membrane. Last report, February 26, 1910: Patient has had no trouble since the operation, and is now feeling much stronger.

(23) Female, aged 33. Illness began fifteen years ago with water-brash after food. Twelve years ago she began to suffer from pain after meals, accompanied by vomiting. She has also had sour eructations. Latterly she has gradually got worse and has lost considerably in weight. Left kidney stitched two years ago, but no improvement followed the operation. She has been under medical treatment continuously for nine months. Pain is referred to epigastrium and comes on directly after food, the vomiting usually about a quarter of an hour later. Pain sometimes extends to *left iliac fossa*, where there is tenderness. Gastric analysis: Stomach empty in morning. Test meal, 200 c.c.—Total acidity, 61; volatile acids, 6·8 per cent.; total chlorides, 0·288; free HCl, 0·007; protein HCl, 0·201; mineral HCl, 0·080. Dr. Fenwick diagnosed gastric symptoms due to appendix. Operation, October, 1909: Duodenum much dilated, no ulcer discovered; after considerable search the cæcum and appendix were found in *left iliac fossa*; appendix very long and contractile, concretion felt at its distal end; internally, appendix filled with *fæces*, concretion at tip, mucous membrane much congested. November 16, test meal, 170 c.c.: Total acidity, 63; volatile acids, 6·8 per cent.; total chlorides, 0·336; free HCl, 0·022; protein HCl, 0·186; mineral HCl, 0·128. Last report, February, 1910: Dr. Young reports that the patient has remained quite well since operation.

INFLUENCE OF APPENDICECTOMY ON HYPERSECRETION.

(8) Male, aged 29. For eight years has suffered from attacks of severe pain in epigastrium, coming on one hour after food. Pain lasts about one hour and is greatly aggravated by solid food, especially meat. The attacks come on at intervals, and usually last a week or two. During the intervals he is free from pain, but suffers from a feeling of discomfort after food. Gastric analysis: Twelve hours after lavage and milk, 50 c.c. fluid in stomach; acid, containing much mucus, and slightly bloodstained. Test meal, 132 c.c.—Bad motility, thick ochre colour, much mucus; total acidity, 85; total chlorides, 0·397; free HCl, 0·025; protein HCl, 0·240; mineral HCl, 0·131. Operation, June, 1908: Stomach and duodenum dilated, no ulcer discovered. Appendix: Some old adhesions, bulbous towards distal end, and two marked constrictions; internally the appendix was markedly constricted in two places, it contained *fæces*, and its coats were thickened. Microscope: Chronic inflammation. After the operation the stomach was invariably empty in the morning, and, although several attempts were made to get a test meal for analysis, the motility of the stomach had so improved that the most that could be recovered was 20 c.c. On several occasions nothing could be recovered at all one hour after the test meal had been given. Last report, November, 1909: Patient had some occasional

discomfort after food for two months after his operation, but since then he has remained perfectly well.

(14) Female, aged 37. Ten years ago severe pain in abdomen, and hæmatemesis; said to have gastric ulcer. The pain has continued on and off since, is referred to epigastrium, and usually comes on about eleven in the forenoon. For the past three months she has been much worse, suffering from severe pain coming on one hour after meals. She has often made herself vomit to relieve the pain. She has also had sour eructations after food, and much flatulence. Ten hours after lavage and milk, 30 c.c. yellowish acid fluid in stomach. Gastric analysis: Test meal, 210 c.c.—Some mucus, fair motility; total acidity, 75; total chlorides, 0.350; free HCl, 0.018; protein HCl, 0.233; mineral HCl, 0.098. Operation, April, 1909: Duodenum dilated, stomach very large, no lesion of stomach or duodenum. Appendix tightly bound down by dense adhesions, distal end bulbous, and contained a faecal concretion. Mucous membrane much inflamed, and considerable hæmorrhage in places. Six weeks later: Stomach empty in morning. Test meal, 124 c.c.—Good motility; total acidity, 72; volatile acids, 4.8 per cent.; total chlorides, 0.336; free HCl, 0.018; protein HCl, 0.260; mineral HCl, 0.058. Patient seen February, 1910: She has been at work since June; feels ever so much better; has had no more pain or vomiting, but still feels weak.

HÆMATEMESIS.

(13) Nurse, aged 25, patient of Dr. Fenwick's. Quite well until December, 1907, then began to suffer from indigestion. February 1908: Severe hæmatemesis; states that she vomited 50 oz.; seven attacks of hæmatemesis within five days. She was treated with horse serum, had rectal feeding for three weeks, and was in bed for three months. She has never been able to eat meat since that date. In May, 1908, she began to suffer from pain in the epigastrium, coming on about twenty minutes after taking food. Since that time she has seldom been free from this pain, and she has had attacks of vomiting at intervals. The pain is referred to epigastrium, and usually comes on from twenty minutes to half an hour after food. The pain gradually gets less, but usually recurs from two to three hours after taking food. If she goes from six to seven hours without food, she has acute pain. Gastric analysis: In stomach, ten hours after lavage and milk, 50 c.c. light-green fluid. Test meal, 130 c.c.—Total acidity, 70; volatile acids, 9.6 per cent.; total chlorides, 0.329; free HCl, 0.015; protein HCl, 0.226; mineral HCl, 0.088. March 8, 1909, operation: Stomach and duodenum explored, no lesion found; appendix bound down by adhesions, and contained a concretion. Microscope: Well-marked chronic inflammation. April 13: Test meal, 50 c.c.—Total acidity, 96; total chlorides, 0.416; free HCl, 0.044; protein HCl, 0.262; mineral HCl, 0.110; volatile acids, 14 per cent. July 8, 1909: Patient very well, has lost all her symptoms. Test meal, 70 c.c.—Good motility; total acidity, 90; volatile acids, 5.5 per cent.; total chlorides, 0.372; free HCl, 0.025; protein HCl, 0.267; mineral

HCl, 0'080. Later report, February, 1910: Patient states that she has remained quite well and never has indigestion except when she eats "really indigestible articles of food." She is at work as a nurse, and "feels strong enough for anything."

(22) Female, aged 42, sent to me by Dr. Stanley Box. Quite well until sixteen years ago; then she had "peritonitis" and was ill for months. The following year she had "ulceration of the bowels." Two years later she had gastritis. Five years ago she had another attack of gastritis with hæmatemesis. Then she remained fairly well until January, 1909, when she was taken ill suddenly with violent vomiting and hæmatemesis, and was in bed for several weeks. Since then she has had three attacks of epigastric pain and vomiting, and she has also had melæna. On September 26 she had another severe attack of pain and vomiting. She had been on milk diet for three months. Seen by Dr. Fenwick, who diagnosed appendicular disease. Pain referred to epigastrium. Marked tenderness in epigastrium, and slight tenderness in right iliac fossa; stomach dilated. Gastric analysis: Test meal, 190 c.c.—Reddish-brown colour; total acidity, 90; volatile acids, 10'4 per cent.; total chlorides, 0'416; free HCl, *nil*; protein HCl, 0'325; mineral HCl, 0'091. Operation, October 7, 1909: Appendix found enormously enlarged, measuring 3½ in. in circumference. Appendicectomy: Appendix contained three large foul-smelling concretions; there was some broken-down blood-clot at the base. Microscope: The appendicular coats are thickened, infiltrated and hyperæmic, the mucous membrane being densely infiltrated. November 11: Test meal, 230 c.c.—Total acidity, 96; volatile acids, 10'4 per cent.; total chlorides, 0'336; free HCl, 0'029; protein HCl, 0'234; mineral HCl, 0'073. February, 1910: Stomach empty in morning; test meal, 210 c.c.—Total acidity, 70; total chlorides, 0'339; free HCl, 0'018; protein HCl, 0'215; mineral HCl, 0'106. Patient is very much better, has no pain, and has had no more vomiting; still has some acidity, and has some tenderness in the wound.

UNSUCCESSFUL CASES.

(16) Female, aged 30. Present illness began at age of 17, with pain in the stomach two or three hours after meals. The pain recurred at intervals of several weeks or months, and very occasionally there was some vomiting. For some time past the pain has been getting more severe. Patient has been losing flesh lately. Some tenderness and pain on pressure midway between umbilicus and anterior spine. Gastric analysis: Ten hours after lavage and milk, 20 c.c. very thick gastric contents, containing bile. Test meal, 242 c.c.—Very thick, much mucus; free HCl; volatile acids, 6'0 per cent.; total acidity, 56. Operation, June, 1909: No lesion of stomach or duodenum; appendix much thickened, some adhesions. Appendicectomy: Mucosa of appendix much thickened, and ulcerated in places. Microscope: The submucous layer is much hypertrophied; the mucous membrane is densely infiltrated and is in process of disintegration. Re-admitted, September, 1909: Patient states that

she was well for several weeks, but latterly the pain has returned; there has been no more vomiting; stomach empty in morning. Test meal, 290 c.c.—Much better motility, less mucus; total acidity, 44; volatile acids, 6.0 per cent.; no free HCl; no peptic digestion in thirty minutes. While in hospital the patient had daily lavage, lost all her symptoms, and put on 7 lb. in weight. December, 1909: Patient has been much better, but says she still suffers from slight pain and discomfort after food; no more vomiting. In February, 1910, patient was re-admitted for observation. While in hospital she slept well, took ordinary diet, and did not appear to have any discomfort.

(20) Female, aged 30, sent to me by Dr. Warren. Thirteen or fourteen years ago patient began to suffer from "risings in the stomach," and seven years ago began to suffer from attacks of vomiting. These attacks have continued on and off ever since, and latterly she says that she had vomited after nearly every meal. Patient has a very chlorotic appearance, skin yellowish-green, nails white, sclerotics pearly white. She complains of abdominal pain, chiefly in epigastrium; worse after food, sometimes relieved by vomiting, sometimes not. Slight rigidity in epigastrium. Gastric analysis: Stomach empty in morning. Test meal, 250 c.c.—Very thick contents, much mucus, bad motility; total acidity, 79; total chlorides, 0.361; free HCl, 0.003; protein HCl, 0.259; mineral HCl, 0.098. Operation, August, 1909: No abnormality of stomach or duodenum; appendix thickened and contained concretion. Appendicectomy: Appendix very long, concretion in middle; mucous membrane deeply congested and petechial in places. Microscope: Much infiltration of appendicular coats, marked ecchymoses in mucosa in region of apex. Patient was quite well while she remained in hospital, and remained well for three weeks after her discharge, then vomiting recommenced. I saw her in December with Dr. Fenwick; she looked much better, although she was still anæmic; there was no tenderness or rigidity of abdomen anywhere. She improved again for a short time and then relapsed. Later note: Patient went to see another surgeon, who diagnosed gastric ulcer, and performed gastrojejunostomy.

Both these patients had such an extreme degree of chronic gastritis that the persistence of symptoms after operation is not surprising. Whether such a condition can be permanently benefited by continuous lavage and dieting is open to question. I have little doubt that in both these cases the appendix was the original cause of the trouble, which was of such long duration (in both cases thirteen years) that it persisted after removal of appendix. In any case, both appendices were so diseased that their removal was certainly advisable. Both patients were sent to me to have gastrojejunostomy performed, and I think the reason of incomplete recovery in the case of the first patient (No. 16) is partly due to disappointment that this operation was not performed.

(17) Female, aged 26. Quite well until eight years ago, then began to suffer from discomfort after food. Treated for indigestion by many doctors; much flatulence. Attended another hospital in April, 1908, and was ordered a belt for movable kidney. Treated by Dr. Fenwick for indigestion since November, 1908. Discomfort, especially after food in epigastrium, sometimes in right iliac

fossa, with occasional attacks of epigastric pain. Always suffers from indigestion. No perceptible mobility of either kidney. As medical treatment had failed, and in view of the fact that she had a small ovarian on the right side, Dr. Fenwick advised removal of tumour and examination of appendix. Gastric analysis: Test meal, 57 c.c.—Total acidity, 68; volatile acids, 12 per cent.; free HCl, 0.022. Operation, July 19, 1909: Ovarian adenoma on right, size of small orange, removed; appendix thickened, peritoneal coat much injected; internally, mucosa much thickened and congested. Microscope: Chronic appendicitis. While in hospital the patient had no further symptoms, but I have heard later that she still suffers from discomfort after food.

(19) Nurse, aged 27. One year ago began to suffer from flatulence and sickness, especially after breakfast. Later, she suffered from epigastric pain, so severe at times that she was incapacitated for work. In April, 1909, she had a severe attack of pain in right iliac fossa, and similar attacks in June and July; continued indigestion. Tenderness on pressure in epigastrium, and to less degree in right iliac fossa. At times she has hyperæsthesia in epigastrium so marked that she cannot bear the weight of her clothes. Gastric analysis: Test meal, 50 c.c. recovered.—Very thick and apparently chiefly ropy mucus; total acidity, 45; volatile acids, 19 per cent.; total chlorides, 0.199; free HCl, nil; protein HCl, 0.096; mineral HCl, 0.103; peptic digestion, none. Operation, August 4, 1909: Appendix very long and bulbous, and contained a concretion. Mucous membrane ulcerated. January, 1910: Patient feels and looks much better, but still suffers from discomfort and flatulence after food. She was readmitted for a short period, and with daily lavage greatly improved and put on weight. This case again is one in which there is such a degree of gastritis that recovery must be slow, if not incomplete. The operation was clearly indicated, as she had had three attacks of appendicitis.

SYMPTOMS.

Pain, varying in degree, is the prominent symptom in these cases. Some of the patients complain of what may be described as severe, continuous discomfort rather than pain, while some describe their pain as severe. In a large majority of the patients the pain follows the ingestion of food, although the interval between the taking of a meal and the onset of pain is very variable. Seven patients stated that the pain came on immediately after taking food, while in twelve instances the interval between taking food and the onset of pain varied from half an hour to three hours. In most cases the pain is referred to the epigastrium, usually to the right of the middle line. Some of my patients stated that the pain radiated downwards to the right iliac fossa, while two with less definiteness said that the pain radiated downwards. In one interesting case the pain radiated to the left iliac fossa (Case 23),

and at the operation the cæcum and appendix were found in the left iliac fossa. This radiation of the epigastric pain to the lower part of the abdomen is, it appears to me, a most important diagnostic symptom, and is one which in my experience never occurs in gastric or duodenal ulcer. Four patients gave a history of "hunger pain." Ten patients complained of sour eructations, and fifteen suffered from vomiting in varying degree. Two patients stated that they made themselves vomit in order to gain relief from the pain.

Hæmorrhage.—Five of the patients vomited blood on one or more occasions. In the case of one patient (Case 13) the amount of blood brought up was said to be 50 oz. One patient (Case 22) while under observation had melæna, and in another case there was a history of melæna. I think there can be little doubt that the hæmorrhage comes from the gastric mucous membrane. In one case in which I opened the stomach the whole of the mucosa was studded with numberless bleeding points. The interior of the stomach was in the condition which has been described as "weeping blood." Probably this condition of the mucosa is due to the irritation of hyperacid gastric juice, or to the continual presence of gastric juice in the fasting stomach. I have seen an exactly similar condition in a case of duodenal ulcer associated with hyperchlorhydria, and I believe that in the majority of cases of duodenal ulcer the hæmorrhage comes, not from the ulcer as is generally supposed, but from the mucous membrane of the stomach. In one of my microscopic sections of the gastric mucosa from a case of duodenal ulcer, blood can be seen escaping between the epithelial cells. I shall refer again later to this bleeding from the stomach without visible lesion, which has been called so aptly "gastrostaxis" by Dr. Hale White. Blood passed by the rectum probably comes from the same source, the blood, instead of, or as well as being, vomited, being passed through the duodenum into the intestine; but in one of my cases (No. 22) it is possible that the blood, or some of it, came from the ulcerated appendix, as at the operation there was a quantity of blood at the apex of the appendix. This case is, perhaps, one of the most striking in my series. The patient had been invalided on and off for sixteen years. She had been treated more than once for gastritis with hæmatemesis. Shortly before she came under my care she had had a severe attack of hæmatemesis, and was kept in bed on a liquid diet for eight weeks. At the operation she had an enormous appendix, measuring $3\frac{1}{2}$ in. in circumference, and containing three large, foul concretions. She had no symptoms referable to the appendix, beyond slight tenderness in the

right iliac fossa. I have seen one case of acute appendicitis associated with hæmatemesis. The onset of more acute abdominal pain and distension led the doctor to think that a gastric ulcer had perforated, whereas the real condition was a perforative appendicitis with general peritonitis. The patient made a good recovery.

Periodical Exacerbations.—A noteworthy feature of these cases is that in many instances there is a periodical exacerbation of the symptoms. This was the case in sixteen patients. None of them were quite well during the interval between the attacks, but suffered from discomfort and flatulence after food. Another striking feature is the long history in a large number of the cases. The average duration of illness in my twenty-four cases is six years, although in one or two of them there was an interval of one or two years between the attacks. Tenderness in the epigastrium, usually to the right of the middle line, is an almost constant feature. In one-third of the cases there was tenderness over the appendix area as well as in the epigastrium.

Sex.—Of the twenty-four patients, twenty were women, the oldest being aged 42 and the youngest 21, the average age being 32. These observations may be tabulated thus:—

TABLE I—ANALYSIS OF SYMPTOMS.

Pain—						
Severe	12 cases
Not severe	8 "
Constant discomfort	4 "
Relation of pain to ingestion of food—						
Immediately after food	7 "
One to three hours after food	12 "
Relation indefinite	5 "
Eructations	10 "
Vomiting	15 "
Hæmatemesis	5 "
Melæna	2 "
Tenderness over appendix	9 "
Exacerbations at intervals	16 "

Gastric Analysis.—I have made complete gastric analyses in seventeen of my cases, and the results are given in Table II. A glance at the table shows that there are considerable variations in the gastric findings. In two of the cases (Nos. 15 and 21) there was marked hyperchlorhydria. In six cases the free HCl differed but little from the normal, while in nine it was absent or nearly so. In these nine cases the findings were those of chronic gastritis, except that the protein HCl was, as a rule, higher than is usual in this condition. In a majority of the cases there is marked increase of the volatile acids. In five of

the cases from 30 c.c. to 40 c.c. of gastric juice were drawn off from the stomach in the early morning ten hours after lavage and taking half a pint of milk. The digestive activity was tested in eight of the cases, and was found impaired in those cases in which free HCl was markedly diminished or absent; in the others it was normal. As a general rule the longer the duration of the symptoms, the greater degree of gastritis shown by gastric analysis.

The Operation Findings.—In fifteen of the cases a diagnosis of gastric or duodenal ulcer was made, and the stomach and duodenum were explored through an incision above the umbilicus. No lesion of the stomach being discovered, the gall-bladder was examined and found healthy. The appendix was then sought for, and, as it showed evidence of disease, was removed, usually through a separate incision over the right rectus, but occasionally through the supra-umbilical wound prolonged downwards. In nine of the cases a diagnosis of appendix trouble was made before the operation, and in three of these the stomach and duodenum were also explored to exclude the possibility of ulcer. In sixteen of the cases in which the stomach was explored the duodenum was markedly dilated. Dr. Mayo told me that in these cases he very frequently observed peristaltic waves passing over the pylorus. Personally I noted such waves in seven only of the eighteen cases in which the stomach was explored.

As regards the condition of the appendix, as a rule, judged by its external appearance, it is not extensively diseased. The most common condition is that the appendix is bulbous, somewhat thicker than usual, and, on opening it, a narrowing or constriction is found near the base, the distal side of the constriction being slightly dilated and giving rise to the bulbous appearance. A concretion was found in exactly half the cases. In all of my cases microscopic examination of the appendix showed the existence of chronic inflammation.

In some of the cases the disease of the appendix was more extensive than that described above. In several of the cases there were minute hæmorrhages in the mucous membrane, and in seven cases there was a definite ulcerated area of the mucosa. In one case (No. 22) the appendix was enormously dilated, measuring $3\frac{1}{2}$ in. in circumference, the mucous membrane was ulcerated, and there were three large, very foul smelling concretions in the lumen, as well as some blood-clot. This patient had had hæmatemesis as well as melæna. In two cases the appendix was not obviously diseased externally, but it was very long (in one case 6 in.) and contractile. In both these cases the lumen of the

appendix was filled with faeces, and microscopical examination showed leucocytic infiltration of the mucosa.

There can be no doubt that an appendix which appears on cursory examination to present no abnormal features may be the seat of disease fraught with grave danger to its possessor. I have on a number of

TABLE II.—GASTRIC ANALYSES IN SEVENTEEN CASES OF APPENDICULAR GASTRALGIA.

No. of Case	Total acidity	Total chlorides	Free HCl	Protein HCl	Mineral HCl	Volatile acids (per cent.)	Duration of symptoms	Condition of appendix
7	64	0.343	0.007	0.204	0.131	—	18 mos.	Concretion in appendix; some adhesions; chronic inflammation
8	85	0.397	0.025	0.240	0.131	—	8 years	Two marked constrictions; chronic inflammation
9	64	0.272	—	0.210	0.062	12.8	10 years	Very long; old adhesions; kinked at acute angle
10	30	0.179	—	0.111	0.068	14.0	3 years	6½ in. long; adherent to right ovary; much thickening of coats
11	63	0.310	—	0.211	0.098	—	7 mos.	Bulbous; foul concretion; adhesions
13	70	0.329	0.015	0.226	0.088	9.6	15 mos.	Bound down by old adhesions; concretion
14	75	0.350	0.018	0.233	0.098	8.6	10 years alight, 3 mos. bad	Dense adhesions; appendix bulbous at tip; concretion; hæmorrhages in mucosa
15	69	0.430	0.073	0.255	0.102	6.4	2 years	Concretion; area of necrosis of mucosa
16	71	0.329	0.025	0.231	0.073	8.4	13 years	Appendix thick; mucosa in process of disintegration
17	68	0.321	0.022	0.226	0.073	12.0	8 years	Thickened, adherent to right ovary
18	82	0.336	0.015	0.281	0.040	4.8	3 mos.	Appendix bulbous; mucosa ulcerated
19	45	0.199	—	0.096	0.103	19.0	1 year	Long, bulbous, containing concretion; mucosa ulcerated
20	79	0.361	0.003	0.259	0.098	—	13 years	Very long and thick; concretion; mucosa petechial in places
21	86	0.412	0.043	0.240	0.137	—	18 mos.	Slight adhesions; bulbous; concretion
22	90	0.461	—	0.325	0.091	10.4	? 14 years	Enormously enlarged, containing three foul concretions
23	61	0.288	0.007	0.201	0.080	6.8	15 years	Very long and contractile, concretion at tip; chronic inflammation
24	40	0.299	—	0.179	0.120	5.6	8 years	Very long; bulbous; constriction near base

occasions removed an appendix which might by an onlooker be regarded as "normal," and yet have been able subsequently to demonstrate to those present the existence of disease within. Such an appendix usually presents the following features: It may not be obviously larger than usual, but inspection shows that instead of being a uniform pale yellowish colour, its surface is covered with a branch-work of minute

TABLE III.—SHOWING GASTRIC ANALYSES AFTER APPENDICECTOMY IN CASES OF APPENDICULAR GASTRALGIA.

No. of case	Interval after operation	Total acidity	Total chlorides	Free HCl	Protein HCl	Mineral HCl	Volatile acids
7	3 weeks	64	0.292	0.015	0.174	0.102	—
10	3 weeks	44	0.22	—	1.130	0.09	20.7 per cent.
11	3 weeks	66	0.350	0.011	0.222	0.116	—
13	5 weeks	96	0.416	0.044	0.262	0.110	14.0 per cent.
Same case	4 months	90	0.372	0.025	0.267	0.080	5.5 „
14	6 weeks	72	0.336	0.018	0.260	0.058	4.8 „
15	6 months	71	0.343	0.018	0.270	0.055	6.4 „
16	4 weeks	56	0.321	—	0.186	0.135	5.6 „
Same case	3 months	44	0.295	—	0.183	0.109	5.2 „
18	1 month	70	0.343	0.010	0.233	0.098	—
21	4 weeks	65	0.298	0.018	0.174	0.105	—
22	1 month	96	0.336	0.029	0.234	0.073	10.4 per cent.
Same case	3 months	70	0.339	0.018	0.215	0.106	10.8 „
23	3 weeks	63	0.336	0.022	1.186	0.128	6.8 „
24	3 weeks	68	0.270	0.011	0.193	0.066	8.8 „

blood vessels—evidence of chronic inflammation. Careful palpation may detect that the appendicular coats are thicker than usual. On cutting the appendix open it usually contains faeces or a concretion, the mucosa is congested, sometimes petechial or even definitely ulcerated. Microscopic examination shows the existence of chronic inflammation. However innocent-looking externally, an appendix which contains a concretion, or the mucosa of which is so damaged as to permit of bacterial invasion, is one which may at any time lead to a sudden attack of appendicitis.

THE EVIDENCE THAT THE GASTRIC SYMPTOMS ARE DUE TO THE
APPENDICULAR DISEASE.

The evidence of the connexion between the appendicular disease and the gastric symptoms is threefold. In the first place there is the observation that the majority of the patients are cured by appendicectomy. If this merely meant that the pain or feeling of discomfort was relieved, the evidence would not be of much value; but it implies more than this, because, in addition to relief of pain, those patients who had suffered from vomiting, melæna, or hæmatemesis, were definitely relieved from these troubles. In other words, objective as well as subjective symptoms disappeared after appendicectomy. I have said that the majority of the patients are cured. Of my twenty-four patients, seventeen have been free from symptoms since operation, three have been improved, and four are "not cured." This does not at first sight seem very conclusive evidence as to the benefit of operation. But be it noted that the patients who have not been benefited by operation are those who have had evidence of very chronic gastritis, and that recovery should be slow and possibly incomplete is not surprising. Two of these patients I have had in hospital again, and with rest in bed, careful dieting, and gastric lavage, they have lost all their symptoms; but whether this improvement is permanent it is impossible to say, as both are comparatively recent cases.

Secondly, the close physiological association between the stomach and the appendix is shown by the improvement in the gastric contents as shown by gastric analysis before and after appendicectomy. The number of cases in which such a comparison has been made is small, as it is only recently that I have been making an analysis after, as well as before, operation. Comparing Table II. with Table III., we see that in six of the nine cases in which there was marked gastritis, a gastric analysis was made after appendicectomy, and in five (Nos. 7, 11, 22, 23, 24) of these six cases there was after operation a considerable quantity of free HCl present in the gastric contents. All these patients are now well. In one patient (No. 16), according to the Table, free HCl was present before operation, and absent afterwards. In this case only one analysis was made before operation and I am inclined to think that the analysis was incorrect. At any rate, since operation, the evidence of chronic gastritis has persisted, and this is one of the most unsatisfactory cases which I have had. Two of the patients (Nos. 13 and 22) had a very marked increase of free HCl after operation, the amount of

free HCl being sufficient to justify the use of the term hyperchlorhydria. This marked increase of free HCl after appendicectomy is very curious, and since I have been making analyses in my ordinary appendix cases I have found it so often that I am inclined to believe that it can hardly be accidental, but must be cause and effect. Further, I have observed that this hyperchlorhydria following appendicectomy is not permanent, but gradually disappears. This is well illustrated in two of the cases in Table III. (Nos. 13 and 22), and I have observed the same sequence in other cases in which I have performed appendicectomy during the quiescent period. Possibly this temporary hyperchlorhydria is due to a protective spasm of the pylorus, Nature's attempt to protect the unhealed site of the amputation of the appendix. I say possibly, but I am very sceptical as to whether pyloro-spasm has any connexion with any of the signs or symptoms of appendicular gastralgia. This observation appears to me to afford strong proof of the causal connexion between appendicular disease and gastric symptoms. Incidentally I may point out that, so far as I have observed, this hyperchlorhydria after appendicectomy is symptomless. One patient (No. 13) one month after appendicectomy had more than double the normal amount of free HCl in her gastric contents, and yet immediately after the operation she was able to take milk without pain or discomfort. I kept her on milk and fish diet for three months, and the amount of free HCl had been diminished almost to the normal. Since then she has resumed an ordinary diet, and, in her own words, "she never has indigestion except when she eats really indigestible articles of food." In this case the symptoms were very severe and simulated ulcer very closely, and her recovery has been very speedy and complete, considering her condition previous to operation. Indirectly such cases are evidence against the view that there is such a thing as a "functional hyperchlorhydria." It is not the hyperchlorhydria which gives rise to symptoms, but the organic disease which is the cause of the hyperchlorhydria.

Thirdly, for the past year I have been, as it were, working backwards and cross-questioning the patients on whom I have operated for acute appendicitis as to previous history of gastric trouble. The frequency with which a previous history of gastric symptoms can be elicited is certainly significant. A number of these patients state that for years they have suffered from what they describe as bilious attacks—that is, attacks of vomiting, with discomfort after food in the intervals between the attacks. More frequently the patients state that for a considerable time before the acute attack of appendicitis they have had to be careful

as to their diet, otherwise they have suffered from indigestion. Dr. Soltau Fenwick in his paper has pointed out that before he recognized the connexion between appendicular disease and gastric symptoms, he was acquainted with a peculiar type of hypersecretion in which death frequently occurred from appendicitis. Some years ago I had an interesting case of this type. I operated on a lady who was supposed to have a gastric or duodenal ulcer. On opening the abdomen I could find no lesion of the stomach or duodenum. The symptoms were clear and definite, and, as the patient had hæmorrhage, I thought that an ulcer must exist undiscoverable from external examination of the stomach. Accordingly I opened the stomach—a procedure of which, by the way, I now most strongly disapprove—and explored it from within. I found the whole mucous membrane in a state of extreme congestion, and studded all over with hundreds of minute bleeding points. I accordingly performed a gastrojejunostomy, and the patient was at once relieved of her symptoms, and remained perfectly well for about eighteen months. She then had an attack of acute appendicitis, and narrowly escaped with her life. At that time I did not recognize the association of appendicular disease with the causation of gastric symptoms, but on looking back I have little doubt that the original gastric symptoms were secondary to chronic disease of the appendix. In this particular case gastrojejunostomy was a success in so far as it relieved the patient of her gastric symptoms, nevertheless I am convinced that I made a mistake in that I ought not to have performed gastrojejunostomy, but to have removed the appendix. Some years ago I saw two cases of gastric catarrh in children, due to latent disease of the appendix, which I did not recognize. In one of these the gastric catarrh was attributed to adenoids, and the boy was sent to me by a well-known physician to have his adenoids removed. This was done, but the boy was not a great deal better, and five months later he had a very acute attack of appendicitis. After appendicectomy he rapidly improved, and has since remained well. The other case was that of a girl who for several years had been subject to attacks of gastritis without obvious cause. Later she had definite tenderness over the appendix, and appendicectomy was performed. Since the operation, now six years ago, she has had no further trouble.

FAILURES AFTER GASTROJEJUNOSTOMY.

I have little doubt, from cases I have seen, that some of the failures after gastrojejunostomy are due to the circumstance that gastrojeuno-

stomy has been performed for gastric symptoms when no gastric lesion existed, and the symptoms have been in reality due to appendicular disease. While I was at Rochester, U.S.A., I saw Dr. Mayo resect two gastrojejunostomies which he had performed for supposed ulcer, and which, with characteristic frankness, he admitted that he ought not to have performed, as the real trouble was proved at the second operation to be latent appendicitis, and I have heard that he has since operated on a number of cases in which a similar mistake had been made by others. I have myself resected one of my own gastrojejunostomies, and two which had been performed by other surgeons, in which no lesion of the stomach existed. One cannot repeat the axiom too frequently that no operation should be performed on a clinical or pathological diagnosis, unless such diagnosis be confirmed by the findings on the operation table. In other words, gastrojejunostomy should never be performed except when a definite lesion of the stomach or duodenum exists, and, further, only when such lesion can be demonstrated without the necessity of opening the stomach or duodenum. If an ulcer exists which cannot be discovered without opening the stomach, it is medical treatment which is indicated, not gastrojejunostomy. This is such a simple proposition that it should be self-evident, and yet it is one which is often disregarded; and I confess at once it is one from which in former years I have departed on two occasions, one of which is narrated above, and in both instances I discovered subsequently that I had made a mistake.

Differential Diagnosis.

The chief conditions from which appendicular gastralgia has to be distinguished are duodenal ulcer, gastric ulcer, and gall-stones. Of these, duodenal ulcer is the disease which gives rise to the greatest difficulty in the differential diagnosis. The symptoms produced by duodenal ulcer and by the cases of latent appendicitis to which I am referring are very similar in many respects. There is often the same history of recurrent attacks; both duodenal ulcer and appendicular disease may give rise to hyperchlorhydria, and also to entire absence of free HCl. Even "hunger pain" may be a symptom of appendicular disease. The points which aid us in distinguishing the two diseases appear to me to be: First, in duodenal ulcer the patient is usually free from symptoms between the attacks; in appendicular gastralgia, even between the attacks, the patient suffers from flatulence and discomfort after food. Secondly, the radiation of the epigastric pain towards the lower part of

the abdomen is highly suggestive, if not diagnostic, of appendicular trouble. Thirdly, the existence of tenderness over the appendix sometimes throws light on an otherwise doubtful case. Fourthly, in many cases of appendicular gastralgia the alteration of the gastric contents is not commensurate with the severity and duration of the symptoms. Let me explain what I mean by this. In a number of the cases which I have examined the gastric contents have been little altered chemically. This is not often the case in duodenal ulcer. In my experience duodenal ulcer most commonly produces or is associated with hyperchlorhydria, at any rate in the early stages; later there is often absence of free HCl. Appendicular disease may apparently exist for a considerable time without a marked alteration of the amount of free HCl. The combination of marked symptoms of duodenal or gastric ulcer with a negative gastric analysis is suggestive of chronic appendicular disease. The cases of appendicular gastralgia associated with hyperchlorhydria perhaps present the greatest difficulty in diagnosis. My impression is that hyperchlorhydria secondary to appendicular disease does not give rise to the severe pain which is in my experience usually associated with the hyperchlorhydria accompanying duodenal ulcer. Fifthly, a point on which too much stress should not be laid is that duodenal ulcer is far more common in men; appendicular gastralgia appears to be more common in women.

In gastric ulcer, food, especially milk, often gives relief for the time being, the pain recurring one or two hours later. In appendicular gastralgia the onset of pain is more variable; food, even milk, usually produces pain or discomfort at once. The dorsal pain, so common in gastric ulcer, is not present in appendicular gastralgia. In gastric ulcer the motility of the stomach is not usually impaired unless the ulcer is near the pylorus, in which case there is usually food retention. The pain in gastric ulcer, or duodenal ulcer, is rarely so severe and continuous that the patient has to take to bed. In several of my cases of appendicular gastralgia, on the other hand, the patients have had to lie up on account of the continuous and exhausting character of the pain. Another point which is helpful in distinguishing appendicular gastralgia from gastric or duodenal ulcer is this: The administration of bismuth and alkalis has little or no influence, in my experience at any rate, on the pain and discomfort due to appendicular disease.

The diagnosis from gall-bladder trouble has been dealt with by Dr. Fenwick in his paper, so I do not propose to discuss this further.

How are the Gastric Symptoms Produced?

When we attempt to explain why appendicular disease should produce gastric symptoms, we are entering into the region of speculation. I think I have brought forward evidence to show that there is a close connexion between the appendix and the stomach, but as to the exact nature of this connexion I am not prepared to dogmatize. Dr. W. J. Mayo thinks that appendicular disease causes a protective spasm of the pylorus. Possibly this is the explanation of those cases in which there is hyperchlorhydria. As Dr. Fenwick points out in his paper, the symptoms are those of hypersecretion. Exactly how the hypersecretion is caused we are not in a position to say. All we know at present is that, in some way, the appendix does affect the gastric secretion. Personally, I think that intestinal stasis rather than pyloric spasm is the condition in these cases. The symptoms may be regarded as toxic, the result of the intestinal stasis. This view seems to be supported by the frequency with which the duodenum is found markedly dilated at operation, and by the fact that in a number of the cases the stomach is dilated also. Further, as a rule, the quantity of gastric contents evacuated after a test meal is greater than in healthy individuals, the percentage of volatile acids is usually increased, and, in addition, flatulence and constipation are prominent symptoms.

Gastrostaxis.

In recent years it is becoming gradually recognized that hæmatemesis, accompanied by pain and vomiting, is not pathognomic of ulcer. Operations have been undertaken for hæmorrhage from a supposed ulcer, but no ulcer has been discovered, the source of the bleeding being a general oozing from the whole mucous membrane. To this condition Dr. Hale White [5] has given the name "gastrostaxis," Dr. Bertrand Dawson [1] has termed it "hæmorrhagic gastralgia." The older writers [4] regarded the bleeding as a form of vicarious menstruation, an hypothesis for which there appears to be no adequate justification. The perusal of hospital reports reveals a remarkable difference between the sex incidence of gastric ulcer as diagnosed clinically, and the sex incidence of gastric ulcer as proved by operation and post-mortem examination. Dr. W. P. Herringham [3], in an interesting paper, has called attention to this difference, and illustrated it from the records of St. Bartholomew's Hospital. There can be no doubt that many of the

cases diagnosed as gastric or duodenal ulcer are not gastric or duodenal ulcers at all, but some other condition, and I suggest that it is probable that some at least of the cases of so-called gastrostaxis, and many of the cases diagnosed as gastric ulcer, are really cases of latent appendicular disease.

Indications for Operation.

Lastly, I wish to make my position quite clear with regard to operation. That latent appendicular disease gives rise to gastric symptoms I have not a doubt. The symptoms I have described are in the main those of hypersecretion or the so-called "acid dyspepsia." I need hardly state that I do not for a moment suggest that all cases, or even the majority of cases, of acid dyspepsia require operation. But I maintain that when we have reason to believe that the gastric symptoms are due to appendicular disease, operation as a rule is advisable, provided medical treatment has failed. I hold it as a cardinal principle that in all cases of gastric disorder, except when there is evidence of some organic lesion, such as carcinoma, pyloric stenosis, or hour-glass constriction, before operation be undertaken it must be premised that medical treatment has been tried and has failed. And when I speak of medical treatment I mean not a few weeks of dieting or drug taking, but a course of rest and careful dieting continued during at least six months.

It is the cases in which there are no physical or chemical signs which require careful consideration. In those cases in which, in addition to the gastric symptoms, there is definite and persistent tenderness over the appendix, operation is usually advisable. I confess that I have an old-fashioned prejudice against operating when pain is the only symptom. At the same time, I recognize that there are such cases in which operation is not only justifiable but necessary. I take it that we are all agreed that it is right to operate in cases of so-called "appendicular colic" when such attacks are recurrent. Surely it is equally justifiable to remove the appendix when the patient suffers from recurrent attacks of gastralgia attributable to disease of the appendix, provided medical treatment has been tried and has failed. And in this connexion I would again point out the diagnostic significance of radiation of epigastric pain to the lower part of the abdomen. I would insist, however, that the pain must be definite, and such that it seriously interferes with the patient's enjoyment of life. The principles I laid down in my Hunterian Lectures in 1906, with regard to gastric ulcer, are as true to-day as when they were written,

not only as regards gastric ulcer, but as regards appendicular gastralgia. "If, after six weeks' complete rest on a milk diet, a further period of six weeks on a milk diet, with comparative rest, followed by three months' careful dieting, the patient is not free from definite symptoms, or if, after apparent cure, the patient has a relapse, operation is probably in the best interests of the patient." And, at the risk of repetition, I may perhaps add what I wrote at the same time: "The performance of gastrojejunostomy (and this is equally true of appendicectomy) in cases of vague gastric disorder, unaccompanied by definite organic lesions, cannot but lead to the discrediting of a valuable and, in suitable cases, legitimate operation."

Conclusions.

(1) Appendicular disease may give rise to symptoms which closely mimic the supposed symptoms of gastric and duodenal ulcer.

(2) The prominent symptom is epigastric pain or severe discomfort after food; in many cases there are sour eructations, vomiting, and even hæmatemesis and melæna.

(3) The radiation of epigastric pain to the lower abdomen is very suggestive of appendicular trouble.

(4) Gastric analysis reveals in some cases hyperchlorhydria, in others a normal amount of free HCl, in others a marked diminution or absence of free HCl. As a rule there is an increase of the volatile acids, and, in some cases, evidence of hypersecretion.

(5) Some cases of hypersecretion or acid dyspepsia, and many cases of supposed gastric or duodenal ulcer, are cases of latent appendicular disease.

(6) The evidence that the gastric symptoms are due to appendicular disease is threefold: (a) The majority of the patients are cured by appendicectomy; (b) the influence which appendicectomy has on the gastric contents; (c) the frequency of a previous history of gastric symptoms in those who have an attack of acute appendicitis.

(7) The symptoms are probably the result of intestinal toxæmia due to intestinal stasis. The effect on gastric secretion is, in the early stages, possibly due to pyloric spasm, but more probably to some influence of the appendix on gastric secretion.

(8) Appendicular gastralgia is apparently more common in women than in men.

(9) The important lessons to be learnt from these cases are : (i) That no operation should be performed on the stomach except when a definite organic lesion of the stomach or duodenum exists. Gastrojejunostomy will not cure appendicitis. (ii) That in all operations for supposed gastric or duodenal ulcer the condition of the appendix should be carefully investigated.

Note.—A few days previous to the meeting at which this paper was read Dr. Mayo very kindly sent me the manuscript of a paper written by Dr. Christopher Graham and Dr. Donald Guthrie, based on the operations performed for this condition in the Mayo clinic. Operations have been performed in 115 cases, with the following results :—

Cured, no symptoms	89 cases
Very much improved	7 „
Improved, occasional return of symptoms	13 „
No improvement	6 „
				<hr/> 115

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Surgical Section.

June 7, 1910.

MR. RICKMAN J. GODLEE, President of the Section, in the Chair.

The Present Position and Treatment of Syphilis.

An Address Introductory to a Discussion on the Subject.

By J. ERNEST LANE, F.R.C.S.

As far as our present knowledge is concerned, syphilis is a malady which can only be mitigated, and which does not seem likely ever to be exterminated. Its ravages at the present time are still sufficiently in evidence to justify its being considered a subject worthy of discussion by the Surgical Section of the Royal Society of Medicine, though, in contrast with a quarter of a century ago, they are comparatively trifling, in that the disease is seldom now productive of the terrible mutilations with which we were familiar in the past. Still, though the more grave external and palpable evidences of late syphilis are not so frequently encountered, yet its remote effects, its influence on the nervous system and on many medical and surgical conditions, cannot be ignored. It is a matter of the utmost difficulty to gauge the incidence of a disease such as syphilis, and a glance at hospital records might give the impression that it was on the increase; for example, the attendances of out-patients at the London Lock Hospital in 1908 were 34,649, whereas ten years previously the number was 25,314—a difference of over 9,000 attendances. These figures, of course, only apply to venereal diseases in general, and not to syphilis in particular, but the ratio of syphilis to other venereal diseases appears to remain fairly constant. This increase in the number of cases treated does not necessarily mean an increase in the amount of

syphilis, but may be ascribed to the fact that the public nowadays more fully recognizes its gravity and the necessity for prolonged treatment. More reliance may be placed on the returns issued by the Army Medical Department, which show a steady decrease in the amount of venereal disease in the Army in the course of the last quarter of a century.

Taking it for granted that the extent of syphilis is considerably less than formerly, and that it is of a less virulent type, one is led to inquire into the causes for this diminution, both in its amount and in its gravity, and the conclusion that will probably be arrived at is that it is due partly to a more efficient system of treatment, partly to the better recognition by the general public of the laws of hygiene, of the dangers of the disease, and of the disasters that may occur if treatment is neglected, and partly due to the process of natural immunity which after the lapse of years must show its influence on any disease. One of the best measures of prophylaxis against syphilis is efficient treatment, and it is to this point that my humble contribution to this discussion will be devoted.

There was until recently a considerable difference of opinion as to the exact period when the treatment of syphilis ought to be commenced: some counselled the administration of anti-syphilitic remedies as soon as the diagnosis of syphilis was arrived at; others, recognizing that it was often a matter of the greatest difficulty to make a diagnosis of syphilis from the appearance and feel of the sore, postponed the commencement of treatment until the appearance of some secondary lesion rendered the diagnosis a certainty. There is no doubt that in the past, through errors of diagnosis, many patients who had never suffered from syphilis have been submitted to treatment for that disease, protracted according to the methods of the individual surgeon. But at the present time such a mistake should be impossible, owing to the momentous discovery of the *Spirochæta pallida* by Schaudinn in 1905, and by the subsequent investigations by Wassermann on the serum diagnosis of syphilis. If the chancre exists, the presence or absence of the spirochæte will prove its nature; while if the sore has healed, then the Wassermann test will clear up any doubts.

It is undoubtedly to the material advantage of the patient that treatment should be commenced at the soonest possible opportunity after a positive and not a putative diagnosis of syphilis has been arrived at, and an early recognition of the spirochæte in the discharge from a venereal ulcer is a conclusive proof as to the nature of the disease.

I have seen a sore of only one day's duration, or possibly recognition, in a patient who had exposed himself to contagion and who had been keeping a keen look-out for possible contingencies. This sore was of the minutest dimensions, and might have been a simple abrasion, might have been a commencing chancroid, or might have been herpetic in nature; but on microscopical examination the spirochæte was found in large numbers in the exudation from the sore, and consequently I was enabled to place that patient on anti-syphilitic treatment almost immediately after the appearance of the initial lesion, and long before a diagnosis could have been arrived at before the discovery of the syphilitic organism.

The diagnosis having been reduced to a certainty by the microscope or by the serum test, the next question is as to the most efficient method of treatment to be pursued. The treatment of syphilis by mercury dates back more than 500 years, and, though that drug has occasionally been threatened by therapeutic rivals, it still maintains its position of supremacy, from which it does not at present seem likely ever to be displaced. But though it is universally recognized as the most powerful anti-syphilitic remedy at our disposal, yet there is great diversity of opinion as to the methods by which it should be administered. Personally, I prefer not to limit myself to any one particular method of treatment, and do not consider that one routine plan is applicable to every case of syphilis; but I would prefer to treat every case on its merits, and according to individual peculiarities. Fortunately, in the majority of cases syphilis is a benign disease, and one which is particularly amenable to treatment, and in a simple case there is no necessity to look beyond the time-honoured method of administration of mercury by the mouth in the form of pills or mixture, and most cases can be conducted to a satisfactory conclusion by this, the most easy and most convenient of plans.

But it is not every case of syphilis that will yield to such simple measures, and it is not every patient who is tolerant of mercurial pills or mixtures. One is often called upon to treat some severe manifestation of syphilis which may be threatening the integrity of some important organ, such as the eye, which may be causing progressive mutilation and disfigurement, or which may be involving the nervous system or other regions of equally vital importance. Again, one may meet with cases in which the digestive organs are intolerant of, and rebellious against, any form of mercury administered by the mouth. For such as these there are other means at our disposal which may by

their potency contend with the immediate emergency, and may not in any way interfere with the digestive functions. First and foremost may be mentioned the method of intramuscular injections of mercurial preparations. I need not weary you by enumerating or discussing the relative merits of the legion of preparations which have been recommended for intramuscular injections, but will proceed to the consideration of this plan of treatment. Intramuscular injections may be given in the form of the insoluble or the soluble preparations of mercury, and, of the two, the insoluble salts are undoubtedly the more valuable, for they produce results which are surprising, alike in the rapidity with which they bring under control manifestations which had previously resisted other kinds of treatment as in controlling the subsequent course of the disease.

The two reliable preparations of the insoluble are (1) calomel, and (2) mercury suspended in some fatty basis and usually designated grey oil, though occasionally christened by the name of someone who has devised some modification of the compound originally introduced by Lang, of Vienna. Of these two methods, that of calomel injections is far the more efficient; indeed they constitute by far the most powerful anti-syphilitic remedy at our command. The dosage of the calomel injections will vary according to the condition, the weight, the age, and the sex of the patient, but the quantity usually administered at each injection is $\frac{3}{4}$ gr. suspended in sterilized olive oil or some similar vehicle. A course of twelve injections given once a week is usually sufficient to clear up any urgent symptoms and to check the progress of any rapidly destructive lesion. In appropriate cases the effect of intramuscular injections is most remarkable, and this plan of mercurial treatment is usually successful when all other measures have failed to check the progress of the disease. Amongst the conditions to which this treatment is applicable are cases of malignant syphilis, attended by severe ulceration and entailing disfigurement and mutilation; syphilitic iritis threatening impairment or destruction of vision; cerebral or spinal syphilis; tertiary syphilitic laryngitis and pharyngitis; chronic and inveterate syphilides, resisting the ordinary methods of treatment; chronic glossitis, and that terribly disfiguring condition, syphilitic macrocheilia. For the past four years I have employed calomel injections systematically in my hospital practice, for the reasons that the disease is more rapidly got under control, that the patient's stay in hospital is thereby rendered shorter, and that the subsequent course of the disease is distinctly modified by this early and energetic attack upon the poison. The objections which have been advanced

against this method of treatment, in addition to those which are inseparable from every plan of introducing mercury into the system, such as stomatitis and gastro-intestinal complications, are severe pain, possibly abscess, pulmonary embolism, and sudden and acute mercurialism. It cannot be denied that the pain following upon these injections is occasionally very acute, and it may be prolonged for several days; but this is by no means an invariable occurrence, and in a certain proportion of cases it is a negligible quantity; for instance, I have come across several patients who, immediately after having received their injection, have proceeded to play a round of golf. As regards abscess, I have met with several cases of severely painful and fluctuating nodosities of the buttocks supervening shortly after the introduction of the injection, but with one exception these have been relieved by aspiration and by withdrawal of a collection of chocolate-coloured fluid, and the subsequent introduction of some boracic lotion. Cases of pulmonary embolism I have not so far encountered. Though many fatal instances of this disaster have been recorded in the past, they were due to the direct introduction of these insoluble compounds into the gluteal veins or their tributaries, and must be attributed to neglect of the anatomical position of the gluteal vessels. There is a zone of safety for intramuscular injections into the buttock, and unless this safety zone is departed from I cannot conceive the occurrence of this severe and alarming complication.

By the use of calomel injections a reservoir of mercury is introduced into the system, into which it should be absorbed gradually, but it occasionally happens that these injections become surrounded by an inflammatory zone which for some reason or other may suddenly give way, with the result that a large quantity of calomel may suddenly be released into the circulation, and symptoms of acute mercurialism may supervene.

Another insoluble mercurial preparation which has achieved much popularity is grey oil, consisting of mercury very finely subdivided and combined with some fatty basis. Its action resembles that of calomel, but it is not nearly so powerful, and falls far short of it in efficiency, though it has some compensatory advantage in that the resulting pain is much less severe. Whilst calomel may be employed in the intractable cases, in those in which some serious complications are present, grey oil is more suitable as a routine treatment, and for this reason is more in vogue in military and naval hospitals, where the patients can be kept under constant observation and supervision.

I shall not devote much time to the treatment by injection of soluble

mercurial preparations, for its drawbacks are so obvious, and its advantages so visionary, that the method compares unfavourably with all the other means by which mercury may be introduced into the system. The principal objection to this method is the number requisite to constitute an efficient mercurial course, for the treatment involves a series of from thirty to forty injections administered daily, and consequently few patients will be found willing to submit to it on the grounds of inconvenience and expense; further, there is the constantly recurring pain after each injection, not so severe, however, as after the use of the insoluble compounds. Another familiar procedure for the introduction of mercury into the system is that by inunction, a very effectual means of getting a patient rapidly under the influence of the drug, but one which, in my opinion, does not compare for efficiency with intramuscular injections of the insoluble mercurial salts, and which has certain counterbalancing disadvantages, which have, in this country at any rate, relegated it to a position of comparative obscurity. Its action is prompt, and it leaves the stomach at our disposal for the ingestion of other anti-syphilitic remedies should they be called for; but the same applies to intramuscular injections, and it cannot be denied that the suggestion of inunction is repugnant to the feelings of a large number of patients. The principal objections to the inunction treatment are that the patient is compelled to regulate his habits according to the requirements of the treatment, and has to follow out a régime which must perforce somewhat interfere with his daily avocations. The necessity of retiring to rest with a moiety of the body besmeared with ointment is an insurpassable objection to a sensitive and æsthetic patient. If the skin is very delicate, inunctions may give rise to a troublesome eczema; while if it is more than usually hairy, localized suppuration in the hair follicles may ensue; further, in the presence of extensive cutaneous manifestations of syphilis it is inadmissible. There is also great uncertainty as to the amount of the drug which will be absorbed, and in some cases its action appears to be absolutely inert; finally, it will not commend itself to the large number of patients who are anxious to conceal the nature of their disease from their friends and relations.

Other methods of administration of mercury are at our disposal, such as fumigations, rectal administration, intravenous injections, and the inhalation and consequent absorption of mercury by the wearing next the skin of flannel or cotton wool impregnated with the drug in a state of fine subdivision.

One of the most important and vital questions is the length of time

during which anti-syphilitic treatment should be prolonged; the general impression in this country appears to be that mercurial treatment prolonged for two years is sufficient to guarantee if not a cure, a subsequent immunity from symptoms. Such a supposition is, in my opinion, fallacious and dangerous, as is evidenced by the large number of cases of late syphilis, specially involving the higher nerve centres, with which anyone who is in the habit of treating the disease must be painfully familiar. The ultimate object of every method of treatment which may be employed is to render the patient impregnable to the ravages of late syphilis, to prevent the occurrence of tertiary symptoms, and especially to guard him from the disastrous effects of syphilis involving the cerebrospinal centres, or important internal organs, such as the heart, the lungs, the kidneys and the liver. We often dismiss a patient as cured, and consider that we have been instrumental in eradicating the baneful poison from his system; not hearing of any recurrences, we conclude that we have effected his cure. But only too often symptoms crop up in later years which the patient may not in any way associate with his more youthful indiscretions, and for which he may consult the neurologist, the ophthalmic surgeon, or those eminent in some particular branch of their profession. The late or tertiary manifestations of the disease are constantly coming before our notice both in hospital and in private practice, and they are the result either of the insufficient duration or of the neglect of the treatment by the patient himself. I have for a long time past recommended that treatment should be prolonged over a minimum period of five years, and in the case of those who are of a neurotic disposition, and whose business entails a strenuous and anxious life, I advocate that it should be extended over seven years. Obviously the treatment cannot be continued for all this length of time, but should consist of one initial course extending if possible over a year, then an intermission of three or four months, and then a series of shorter courses of treatment and more lengthy periods of intermission, until the expiration of the number of years deemed necessary for each individual case. Long before the expiration of that time the patient will in most instances have been incapable of transmitting the disease to his wife or to his offspring; but, though he may have attained this point, he is not himself necessarily immune from the occurrence of nerve or visceral syphilis. We are not yet sufficiently conversant with the exact significance of the Wassermann test for syphilis. No doubt the test often remains positive long after the patient has ceased to be a source of danger to others; but, if positive, it

appears to me to be a sufficient indication that the patient will be benefited by further courses of treatment until the test becomes permanently negative.

I shall not dwell at any length on treatment by the iodides, which are of the greatest value in getting rid of certain syphilitic manifestations, though they do not appear to be capable of eliminating the poison of syphilis in the slightest degree. Some patients appear to be unusually susceptible to the action of iodides, and show symptoms of iodism very shortly after the treatment has been commenced. Many modifications of iodine have been suggested by which the disadvantages and discomforts of iodism could be obviated or modified. One preparation which has attracted favourable attention is iodipin, a compound of iodine and sesame oil; this is said to be more efficacious in the form of intramuscular injections than when administered by the mouth, as more iodine is liberated into the system by the former method. This preparation is hardly so popular here as at Continental health resorts such as Aachen, where it is given in enormous doses, such as 3x (ten drachms) of the 25-per-cent. solution daily, until as great a quantity as 40 oz. has been introduced in the space of two months. I have seen cases in which the administration of this substance was productive of excellent results; but, on the other hand, I have seen a patient who suffered from almost complete atrophy of the gluteal muscles in consequence, and another who was leaking out iodipin from many punctures in his buttocks, to his great embarrassment and disgust, and possibly to that of others.

For many years past arsenic has been recognized as a valuable adjunct to mercurial treatment, especially in the form of Donovan's solution, a preparation which is specially indicated in the treatment of late and intractable syphilides. But the amount of arsenic that can be introduced into the system by way of the stomach is very limited, the dose being from $\frac{1}{60}$ gr. to $\frac{1}{15}$ gr., and any larger doses are liable to be followed by signs of arsenical poisoning. In order to facilitate the introduction of larger quantities of arsenic into the system, certain compounds known as the aryl-arsenates have of late years been introduced, and have met with some amount of success and support in the treatment of syphilis. I need not trouble you with details as to the composition of the various aryl-arsenates, but will enumerate those with which we are most familiar in this country. They are atoxyl, containing 27.3 per cent. of arsenic; soamin, with a percentage of 22.8; arsacetin, 21.25 per cent.; and orsudan, 25.4 per cent., to which may be added

another preparation known as atoxylate of mercury, containing 23 per cent. of arsenic and 31·8 per cent. of mercury. Of these preparations, the two with which I am most familiar are soamin and orsudan, both of them products of Messrs. Burroughs and Wellcome, and I have notes of forty-four cases treated by the latter preparation. The dose I used was ten intramuscular injections of orsudan, 10 gr., introduced on alternate days, so that by the end of nineteen days over 25 gr. of arsenic had been introduced into the system. The success obtained by this treatment was at first most encouraging, and certainly it caused the disappearance of the early symptoms of the disease with wonderful rapidity. In very few of the cases were symptoms of arsenical intolerance observed, and these disappeared on prolonging the interval between each injection. But eventually I met with a catastrophe so grave and so disturbing that since that event I have entirely discarded this form of treatment.

A patient, aged 49, an old soldier, was admitted to the Lock Hospital suffering from a typical indurated sore and a papular syphilide on the face and trunk. He was given nine injections of orsudan 10 gr. on alternate days; on the day after the seventh injection he noticed general fogginess of vision in both eyes, which gradually increased, but he omitted to mention this fact until the day after the ninth injection. Then, on examination, there was found to be tremor of both eyes, and difficulty in counting fingers held up at a distance of 12 in. in a strong light. On ophthalmoscopic examination both disks were found to be pale and indistinct in outline, with the vessels of normal calibre. A very careful general examination showed absence of any signs of arterial degeneration, the kidneys were apparently perfectly sound, and the physique and general health were perfectly good. He was treated with large doses of strychnine hypodermically, and subsequently with calomel injections. Examination of the eyes four months later showed a perception of hand movements at a distance of 4 ft., equal in both eyes; both disks were markedly atrophic, the atrophy not indicative of a previous neuritis, whilst there was well-marked pigmentary changes at both maculae suggestive of inflammatory or degenerative changes.

Shortly afterward I met with another very similar case of a patient, aged 46, who had been treated by a private practitioner with injections of soamin. He had received ten injections of soamin, 5 gr. on alternate days; after the third injection he noticed some haziness in vision more marked in the right eye; after the sixth injection the impairment of vision became more marked, but he could still see objects at a distance, though definition was not good. After the first course of soamin all signs

of syphilis had disappeared, and treatment was stopped for two months. He then was given five further injections, this time of 10 gr. of soamin on alternate days, but then the vision became so obscured that he became alarmed, and declined any further injections. Assuming that his ocular symptoms were attributable to syphilis, he applied for and obtained admission to the Lock Hospital. He then had no signs of syphilis, his general health was good; there was no sign of arterial disease, and both skin and kidneys were satisfactorily performing their functions. On examination of the eyes there was found to be perception of hand movements at 6 in. in the right eye and 18 in. in the left; both disks showed typical primary optic atrophy, and there was no alteration in the retinal vessels and no alterations in the maculæ.

I felt it my duty to draw the attention of the profession to the dangers which might ensue from the employment of these preparations, and communicated with one of the medical journals on the subject.¹ My letter was followed by various comments—some unfavourable and accusing me of creating an unnecessary panic, others agreeing with me that my warnings were perfectly justified. In confirmation of my warning, I learn that the treatment has been discontinued by such authorities as Professors Neisser and Finger, owing to the occurrence of similar mishaps. It cannot be denied that the results of this treatment have been satisfactory in some of our military hospitals, but opinions as to the value of the aryl-arsenates differ widely amongst the officers of the Royal Army Medical Corps. Whereas some regard it as a prophylactic against any further developments of the disease, a contrary opinion is expressed by a memorandum issued in January of this year by the expert Committee on Venereal Disease in the Army to this effect: "That the utility of these preparations as a prophylactic against syphilis has not been proved, and the Committee does not recommend their administration for that purpose."

It seems to me to amount to this: that the aryl-arsenates do, in a certain number of cases, considerably modify the evolution of the early symptoms of syphilis, but they are just as liable to be followed by relapses as is the treatment by mercury, which also appears to be more reliable; that we have, in the various methods of administration of mercury, a perfectly satisfactory remedy for the treatment of the disease, and one which, with ordinary care, is at any rate free from danger. In presence of a disease such as syphilis, which, in the vast majority of cases, is amenable to mercurial treatment, the effects of which, though

¹ *Brit. Med. Journ.*, 1910, i, pp. 599, 725.

slow in many cases, are, for the most part, sure and safe, I cannot think we are justified in having recourse to remedies such as these arsenical preparations, which are capable of causing such catastrophes as those I have already alluded to. But with a disease such as trypanosomiasis, which is almost certainly fatal unless drastic measures are resorted to, the employment of these remedies is not only justified but is imperatively called for, since the possibility of blindness is better than the certainty of death.

Dr. ALEXANDER FLEMING said that his contribution to the discussion would not deal directly with the treatment of syphilis, but he would try to show in what ways the laboratory worker could help the surgeon in the treatment. In the first place he would deal with the diagnosis of syphilis, for, as Mr. Lane had strongly emphasized, early diagnosis and the early commencement of treatment were most important factors in the successful conduct of the case. In most cases clinical observation was all that was required, but there were always cropping up cases where the signs were not clear, and some further aid was necessary. The laboratory diagnosis of syphilis could be divided into two parts:—

- (1) Finding the *Spirochæta pallida*.
- (2) Serum test (Wassermann's reaction).

The spirochæte was most readily found in the primary stage, and in some of the lesions of the secondary stage, as in the other lesions, either the organism was present in very small numbers or the disease was situated in some inaccessible region. It was in the primary stage, however, that the diagnosis by this method was most valuable, as it was in this stage that the clinical evidence was least convincing. The methods which had been used in the past for finding the organism were: (1) Staining the organisms by one of the various means recommended; and (2) cutting sections of tissues which had been impregnated with silver. These methods had now been practically given up in favour of the two more recent methods, which were now used almost exclusively. These newer methods were the dark-ground condenser and the Chinese-ink methods. The dark-ground condenser had largely been used in the last year or two, and probably it furnished the most accurate knowledge in some cases, as it had the advantage over all other methods that it demonstrated the characteristic movements of the spirochæte. It had the disadvantage, however, that it required a special condenser, which one had to fit into the microscope when one wanted to look for the spirochæte; and that it required a certain amount of skill and experience to be able to get a good picture, so that in the hands of any other than the

trained microscopist it was much less convenient than the Chinese-ink method (Burri's method). The technique of this was childishly simple, and it required no special materials except a bottle of Chinese ink (which could be readily obtained at an artist's material shop) and a microscope. (He then demonstrated the method.) The spirochaetes appeared as clear spirals on a black field, and could be found very rapidly and with great ease. The disadvantage was that the movements of the organisms could not be seen. By one or other of these methods one could quickly arrive at a definite diagnosis in practically all cases of primary syphilis.

As regards the serum reaction, it was of least value in the primary stage, although even in this stage of the disease positive reactions were obtained in about 65 per cent. of cases. In primary syphilis, then, a negative reaction was of little value in excluding syphilis from the diagnosis, whereas in the later stages a negative result was of almost as great value as a positive. In the secondary stage, out of about 100 cases tested he had found only one, or possibly two, in which a positive reaction had not been obtained. In tertiary and congenital syphilis also the reaction was positive in practically every case. In parasyphilitic affections there was a marked difference between general paralysis, which gave 100 per cent. of positive reactions, and tabes, in which the test only succeeded in 65 per cent. to 70 per cent.

In making these observations he had used, not Wassermann's original method, but a modification which he had demonstrated before the Clinical Section of this Society in May of last year.¹ This modification was the same in principle as Hecht's method, and as certain objections had been raised to Hecht's method, they would equally apply to the method he (Dr. Fleming) had used. As it was possible that these objections would be brought up later in the discussion, he would briefly allude to them. The chief criticism of the method (which was urged mainly on theoretical grounds) was that it was possible to obtain by it a positive reaction in some cases which were not syphilitic. This objection he thought to be absolutely without foundation. He had tested over 500 persons who were supposed not to have syphilis. Some of these were patients attending the hospital for various diseases; others were workers in the laboratory, and visitors. Of these 500 cases, only some twelve gave positive reactions; and on inquiring into the history of these, facts were elicited which made it probable that there was a syphilitic infection. In no other disease than syphilis had he found positive reactions, with the

¹ *Proceedings*, 1909, ii (Clin. Sect.), p. 220.

exception of 3 cases of leprosy and 1 case of trypanosomiasis. Scarlet fever, pneumonia, and various other diseases which have been at times described as giving positive reactions, had all yielded negative results. Possibly the reason why some observers had obtained positive reactions in non-syphilitic cases was that they used either an antigen which was unsuitable for the test, or that they had used the antigen too strong, as by using too strong an antigen it was quite easy to deviate the complement of normal people.

The latest figures, comparing the two methods, are those of Hecht,¹ which show that of 142 normal people tested, one gave a positive reaction by both Wassermann's and Hecht's method (this man was in all probability a syphilitic who denied infection), while of the cases of known syphilis only one gave a positive reaction with Wassermann's method and failed with Hecht's; whereas 39 failed to show the reaction with Wassermann's technique and gave positive reactions with Hecht's. This showed that the simpler method was also the more delicate. Mr. McDonagh, in an article in the *Lancet*,² recently stated that the probable reason why the test did not come off in every case of florid secondary syphilis was that in heating the serum for half an hour before the test was commenced (as is necessary in Wassermann's method) the complement was converted into complementoid, which possessed the combining properties of complement without its active properties. If this is so, then it placed Wassermann's method at a great disadvantage as compared with Hecht's, in which the serum was not heated.

With regard to the effect of treatment on the Wassermann reaction, there seemed no doubt that treatment by mercury sufficiently energetic, and prolonged for a sufficiently long time, destroyed the serum reaction. If treatment was stopped too early, then after an interval of a few months the reaction would return, but if the treatment was sufficiently prolonged the reaction seemed to disappear for good. The reaction had not been long enough in use for anyone to follow any one case for a sufficient length of time to show that after a long interval the reaction might not return; but from an examination of cases repeatedly at varying times after treatment, it was held that if it did not return within a year after treatment had ceased the case could be regarded as cured.

He had been making observations for some months past on the effect of treatment on the serum reaction, and, as the cases came from different

¹ *Zeitschr. für Immunitätsforsch. u. exper. Therap.*, Jena, 1910, v, p. 572.

² *Lancet*, 1910, i, p. 922.

sources and had been treated in different ways, he had been in some ways able to compare these different modes of treatment. The number of cases treated by any one method was, however, somewhat limited, so that further work with more cases might modify his conclusions. Three methods mainly had been compared—injections of grey oil, inunction, and oral administration of pills—and they seemed to be efficacious in the order named. In cases treated with grey oil the reaction disappeared in about 40 per cent. after only twelve months' treatment, whereas with pills half the cases tested after three years' treatment still gave a positive reaction.

He (Dr. Fleming) suggested that in the treatment of syphilis the surgeon should continue the administration of mercury for such a period as he thought advisable, and after a short interval, during which no mercury was taken, the reaction should be taken. Should this prove positive, then treatment should be recommenced; but should it prove negative, the serum test should be repeated after an interval of six months or a year. Should this again prove negative, then the case might be regarded as cured, although, if the patient wanted further reassuring, the test might be repeated at long intervals.

Major H. C. FRENCH, R.A.M.C., wrote as follows: Our position in regard to the treatment of syphilis is, I am afraid, too often influenced, unless the cases are in hospital, by the convenience of this or that individual. To treat syphilis adequately, and so keep the disease under effectual control, it appears to be absolutely essential to recognize it in the earlier and more remediable stages. If the *Spirochaeta pallida* is demonstrated, or if the classical sign of induration exists in the chancre and in the adjacent lymphatic glands, we should, I consider, vigorously attack the disease by *judicious* mercurial treatment. The Wassermann serum test cannot be implicitly relied on in the primary stage. I do not agree with those persons who counsel a waiting policy in all cases until the rash is present. The rash, in my experience, does not, as a rule, appear until fourteen to sixteen weeks from the date of exposure to contagion. Delay for this period in commencing treatment is unjustifiable in probably 90 per cent. of cases, and markedly aggravates the disease. In a small minority, however, where induration of the chancre and discretely enlarged lymphatic glands are not marked, we must perforce wait for more definite evidence, such as rash, &c.

A course of mercurial treatment before the date of onset of rash is, I consider, worth two courses later as regards the ultimate beneficial

effect on the disease as well as in its effect in guarding against parasymphilitic affections. As regards *duration* of treatment, a period of two years with intervals between courses is my usual rule—where inunctions or hydrargyrum cum creta with opium have been used—but when insoluble preparations of mercury, such as grey oil, are employed, a third year's course of treatment is often necessary to dissipate the remaining external evidences. Relapse or accentuation of manifestations whilst actually under treatment is, in my experience, rare with the inunction or hydrargyrum-cum-creta-with-opium methods in hospital, but quite commonly observed under treatment by insoluble preparations of mercury. An increase of inefficiency in hospital consequently results, which is important in the Army from the point of view of the State.

Inunctions of unguentum hydrargyri form the routine method of treating syphilis in the French, German, Italian, Bavarian, Hungarian, Swedish, and Danish armies; also at Aix and Wiesbaden and Unna's and Pontoppidan's clinics on the Continent, and Harrogate in this country. This valuable method is now receiving much more attention in the British Army. I employ it in the majority of cases in doses of $\frac{3}{4}$ dr. to 1 dr., with a daily hot bath (preferably sulphur), and thirty to fifty rubbings form a course in early syphilis, according to the indications of the particular case, and controlled by the weekly weight. The patient must be seen daily by the medical man, and be kept in hospital. A mouth wash of acetate of alum is used every two to three hours, and all carious teeth extracted or attended to carefully, and tooth powder used twice daily. All the minutiae of technique must be rigidly adhered to in order to attain good results, and the patient well fed with milk and eggs in addition to ordinary food.

The rapid disappearance of induration in the chancre and glands is the best test of progress, and where this has occurred the disease can be well kept in hand by the avoidance of smoking and local treatment, if mucous patches develop, until the second course of treatment, some six weeks later, by inunction or other methods. In the treatment of mucous patches in the mouth, ulcers on the tonsils, and laryngeal affections, the value of soft, non-irritating food such as meat, stews, &c., cannot be too strongly insisted on.

The Wassermann-serum-diagnosis test, according to the evidence of Captain Gibbon, R.A.M.C., recently working on cases in my clinic in Malta, is by no means always conclusive before the development of the constitutional signs of rash and generalized glandular enlargement. In several cases (where treatment was not employed) there was well-

marked induration of the chancre and proximal lymphatic glands, the Wassermann test was negative, and syphilis supervened. In other instances, in which there was no clinical evidence of syphilis and no treatment employed, the Wassermann test was *positive*, and syphilis did not supervene later. The technique employed was that recently outlined by Captain Gibbon in the *Journal of the Royal Army Medical Corps*, where he gives 98 per cent. of *positive* results in cases with well-marked clinical evidence of the disease, the majority of which I had recently placed on the syphilis register. The Wassermann test gave valuable results in parasyphilitic cases. The result was frequently positive after two to three years' mercurial treatment, and this is in consonance with the views recently expressed by Harrison independently working at the Rochester Row Military Hospital, London, and quoted in the *Journal of the Royal Army Medical Corps*, March, 1910. The inconclusive nature of the Wassermann-serum-diagnosis test in the *early* stages of syphilis before secondary (constitutional) manifestations of the disease have appeared, is also recently outlined in the *Epitome of British Medical Journal*, and also on May 7, 1910.

The foregoing and following facts were dealt with in detail in an article in the *British Journal of Dermatology* in November and December, 1908, and the *Lancet* of September 25, 1909, and January 8, 1910, so I do not dwell at length on them. The average manifestations in syphilitic cases are mild, and hence new remedies are very apt to be unduly exploited, to the possible detriment of older, well-tried, and probably better methods.

The marked reduction of syphilis in the British Army in recent years has mainly occurred in India, not from the use of insoluble grey oil, but as the result of the control of diseased men and women under the excellent and moderate provisions of the Cantonment Code of October, 1897, as well as by systematized hospital and out-patient treatment. There has been a corresponding reduction in gonorrhœa and non-infecting chancres. I understand that in London there are less than 100 beds for the treatment of venereal disease in civil hospitals, whereas in Paris there are 2,000. Consequently disease is unduly and quite unnecessarily spread, and facilities in England for obtaining the "*connaissance approfondie*," so requisite for adequate treatment, and insisted on by the Brussels International Conference of 1899, are unduly curtailed.

Most persons will admit that iodide of potassium is a valuable drug in treating certain phases of syphilis and its utility more obvious the

longer the period from the primary evidence of the disease. I do not think it should be used to the exclusion of mercury, but to supplement it. My experience of soamin and arsacetin is that they exert a tonic action in the early phases of the disease, and commonly cause a temporary gain of 5 lb. to 7 lb. in weight amongst cases treated in hospital; but in many instances severe relapse has occurred with rapid loss of weight, and fits of an epileptiform character have ensued.

The risk of blindness from optic-nerve atrophy has been not infrequently recorded by other observers after the use of soamin and arsacetin in treating syphilis, as well as with atoxyl in treating sleeping sickness. The latter drug has caused complete blindness in fifty-four cases and partial blindness in 136 cases.¹

In London hospitals I would suggest the provision of beds and the establishment of professorships, as in the United States, for this particular branch of medicine. In the Army, with the marked reduction that has occurred in the past ten years, venereal diseases even still cause 27 to 30 per cent. of the "constantly sick" in hospital from all diseases in all parts of the world. What is true of the Army would no doubt equally apply to the relative incidence in civil practice. A Royal Commission reported in 1889 that the cost to the State of maintaining and educating 7,000 blind persons in the United Kingdom, whose blindness resulted from a preventable disease, ophthalmia neonatorum (due to gonorrhœa), was £350,000 a year.

I would also suggest the appointment of a committee of medical men in England to collect definite views on all these various points on a somewhat similar basis to the valuable Advisory Board Reports on Venereal Diseases in the Army published in 1905-6. Having collected these views, to memorialize Parliament to adopt suitable measures whereby this disease be not so largely spread in view of the immense cost to the country in maintaining lunatic asylums, hospitals, and syphilized persons in poor-house infirmaries and prisons. In some countries in Europe medical men, lawyers, clergymen, and others combine to form societies to combat this evil from the medical, social, and moral point of view, and with good results.

In conclusion, one may add with Professor Fournier, of Paris, in imitation of another proverb, "that the fear of syphilis is the beginning of wisdom"; and re-echo that fine thought of Pasteur's, "that where good is to be done, duty only ceases when we no longer have power to achieve more or to do better."

¹ Vide *Brit. Med. Journ.*, 1910, i, p. 1097.

Captain L. W. HARRISON said that Mr. Lane had covered the ground from a clinical point of view so completely that he had himself very little to say from that standpoint. He would only refer to the treatment of syphilis from the standpoint of administration and pathology. He would have liked to hear a little more on the question of prophylaxis. Mr. Lane said there had been a great decrease in venereal diseases generally in the Army of late years. He thought most Army medical officers would agree that that was not due to any one particular treatment, but more to the work which was being quietly done by medical officers, in spite of a very hostile party at home. He mentioned the subject in the hope that something might be done by the civilian members of the profession to educate public opinion more in this direction. If officers in India were known to recognize women of a certain class, or deal with them in any way which would prevent them conveying the disease to soldiers, questions were asked in Parliament, and they ran the risk of losing their commissions. That was very wrong, and much might be done to educate public opinion at home as to conditions in India. There should be no such risk to officers because they tried to prevent the spread of the disease. With regard to the various methods of treatment, it had been found most practicable in the Army to use the injection of some insoluble salt of mercury. The injection method was not compulsory in the Army. The majority of the officers in the beginning received the innovation with prejudice and disfavour; they were enamoured of the mouth treatment at that time. But injection had gradually won its way, and it was found to be the most certain method of insuring that the patient actually got the mercury. With regard to the mouth treatment, he would mention what was said by Staff-Surgeon Adams in the case of the Navy, where the mouth treatment was more in vogue than in the Army. That gentleman said that they collected the sweepings from a certain place and filled about half a bucket with hydrargyrum-cum-creta pills which were supposed to have been swallowed by various patients in the Navy. With regard to the use of calomel cream, his experience in reference to that was similar to Mr. Lane's—that it was a valuable method, especially in the cases which required urgent treatment. But he was surprised to find that Mr. Lane could give twelve injections of calomel cream. The experience at Rochester Row was that patients were generally intolerant of more than three injections of it; they had to go on with grey oil or one of the new preparations, creocamph mercurial cream. Inunctions were, from the Service point of view, impracticable. In a large number of

cases it would be impossible to rub men as it ought to be done, but it was recognized that inunctions should be used in certain cases. Rubbers were trained at Rochester Row, and inunction was given where it was deemed advisable. For all cases to be rubbed, considering the time taken for each case, was impracticable, and therefore injections were regarded as more convenient.

It was mostly agreed that the earlier treatment was commenced, the better the ultimate result. But he regarded it as absolutely wrong for treatment to be commenced on mere supposition, especially as more exact means of diagnosis were now available. The disadvantage of delayed diagnosis did not compensate for subjecting patients to treatment on insufficient grounds. He had recently seen cases of severe nervous disease where the patient or his doctor originally thought he had syphilis, and mercury had been given to see whether it would act or not. In such cases confidence was often lost in the original diagnosis, treatment was suspended, and nothing further was heard of the case until ten years later tabes developed. Now that there was the serum diagnosis cases of doubt should not be treated till some laboratory test had been instituted to settle the doubt. With regard to the treatment and control of syphilis, he agreed with Mr. Lane that two years was not sufficient time. At Rochester Row after two years' treatment the case was sent to him (the speaker) to see if the Wassermann reaction was positive. If it was, he was not struck off the register. If the reaction was negative, the man was asked to come back in three months' time, and his blood was tested again. That could not yet be carried out throughout the Army, but he hoped that it would be eventually. He had tested a large number by the original Wassermann technique, which he thought was the most reliable, especially from the point of view of diagnosis, though he agreed it was not so delicate as Hecht's and Fleming's modification of Hecht's. He had made experiments to find out what was the effect of heating, and he found the length of time during which the serum was heated made a good deal of difference. By heating for not more than ten minutes he got more delicate reactions than by following the original method of heating for half an hour. He carried out the tests in a roughly quantitative manner, which gave him more information than by the simple test. He tested whether the patient's serum would absorb the standard amount of complement, and also double that amount. He had made notes of every case of syphilis he had thus tested, and had tried to correlate the whole of his results with the treatment. He got 93.5 per cent. of positive reactions in

untreated cases, taking them as a whole and including primary cases, from the fifteenth day. After one course it dropped to 83·6 per cent., and there was a steady drop, till after six courses it was 34·6 per cent. The latter figures, however, were based on a rather small number of cases, and he should place the percentage of positive results after six courses at about fifty. After eight courses the percentage had dropped to 21. But still there was an important percentage of positive reactions after two years' treatment; on those grounds he agreed that treatment should be prolonged for a longer period than two years, and he hoped that would become the rule in the Army.

With regard to the particular method of serum diagnosis to be adopted, Dr. Fleming mentioned that his and Hecht's method were reliable. From the point of view of controlling treatment, he (the speaker) thought such modifications all right; he himself employed a modification—Stern's—in conjunction with the original, but from the diagnostic point of view there were a number of fallacies, and Dr. Fleming would agree that there must be fallacies in a method in which the constituents were not constant. One should rely for diagnosis on the method in which the reagents were most constant. He had seen a positive result by Fleming's method in a normal case, and he would be sorry to say it was an error in technique, because it occurred to a worker who had practically lived in a laboratory during the last fifteen or sixteen years. The control came out all right; he saw it himself: it was a clean result, and it was not a case of too strong antigen. He could see nothing for it but a fault in the method or in the modification; it was likely to occur when the patient's serum was somewhat short of complement, but not entirely deficient. The result in testing such a serum was that the extract was sufficient to turn the scale against hæmolysis in the tube containing it, whereas there was no extract in the control tube and hæmolysis occurred.

Mr. J. E. R. McDONAGH: *Prophylaxis*.—During recent years much legislative ingenuity has been expended on the Continent by the State control of prostitution. The result has been disappointing, for syphilis seems to have increased on the Continent. The reason for this is a simple one. While State control has undoubtedly rendered syphilis less common amongst professed prostitutes, it has increased the number of unregistered, and therefore uncontrolled, women, among whom syphilis has become the rule rather than the exception; and for such an Act to bear fruit, syphilis in men should be equally notifiable. Without having

State control of prostitution there seems to be absolutely no reason why syphilis should not be included under the Contagious Diseases Act.

Serotherapy.—Serotherapeutic measures and preventive treatment by means of injections of the serum of animals, which had previously become infected with the syphilitic virus, have proved hopeless. Neisser injected, intravenously, a syphilitic in the primary stage with the serum of a patient who had had syphilis, but with no result. Metchnikoff and Roux, having proved *in vitro* that the serum of apes which had previously been infected with syphilis possessed some parasitocidal power, tried to produce a prophylactic active immunity by obtaining a virus weakened by the passage through several species of apes, which possessed a certain natural resistance against syphilis; but, unfortunately, the results proved fruitless. Kraus obtained a vaccine from the maceration of chancres and injected it subcutaneously in the primary stage of the disease; but although Kraus's reports are favourable, other observers have failed to corroborate them.

Excision of Chancre.—The treatment of syphilis should be begun the moment the disease has become diagnosed—as Mr. Lane has in his address so strongly advocated—since we have every means of diagnosing a suspicious sore the moment the patient's attention has been drawn to it. Experiments proving that the inguinal glands are affected before the patient comes up for advice have made most observers look upon excision of the chancre as being unnecessary, but they fail to realize that a chancre left may act as a fountain from which fresh organisms continually flow; and that a chancre may relapse, that papules and gummata may occur at the site of the initial lesion, and starting treatment at once causes the chancre to heal over quicker, and therefore diminishes risk of infection. I know of two cases in which the chancre had been excised after the discovery of the specific organism about two weeks after its appearance. The patients were not treated. A Wassermann's reaction was carried out every three months for two years—always with a negative result—and no further symptoms of the disease appeared. To my mind there is no doubt that when a chancre can be easily excised, it should be, as I feel quite sure that the future course of the disease is affected thereby.

Treatment and Wassermann's Reaction.—The influence of treatment on the Wassermann's reaction is undeniable, but the treatment must be mercurial, as potassium iodide has no influence on converting a positive into a negative reaction. Mercury given by inunctions has far and away the greatest influence in changing the reaction, it being practically the rule after a course to obtain a negative reaction, which later may become

positive, depending upon the number of courses given. Owing to the fact that syphilis may run a mild as well as a severe course, it will be by the Wassermann's reaction that we shall be able to gauge as to whether a patient requires more treatment or not. This is, in my opinion, the greatest argument in favour of the intermittent over the continuous treatment. We have not yet arrived at the stage when one can say positively as to whether a patient should receive more treatment or not, but it is a very safe rule to go on to say that a patient is cured when his blood gives a negative reaction three, six, and nine months after his last taking mercury. Such a course will not increase proclivity towards getting nervous manifestations, since such diseases are noted for the amount of anti-substance present in the patient's serum. Injections play a subordinate rôle to inunctions, and it may require several before any change in the reaction is noticed. Pills as a routine and continued treatment hardly come under consideration except to be doomed: first, because they have little or no influence in changing the reaction; secondly, because they upset the patient, increase the intra-buccal lesions, and predispose towards carcinoma of the tongue. Over and over again I have noticed how bad patients' mouths and throats get when they are taking pills; it is not due to a mercurial overdose, since alleviation sets in the moment injections or inunctions are given. While on the Continent I was struck with the rarity of bad tongues (including carcinoma), although syphilis was so much commoner than at home. One cannot go into a surgical clinic in London, however small it may be, without seeing several cases of bad tongues, which get worse the further pills are pushed; not infrequently patients have asked to have injections rather than pills, owing to the bad state the mouth gets into when taking the latter. One of the greatest arguments in favour of treating the patient as early and as vigorously as possible is shown by the fact that, when a case has progressed to a certain stage, so as to produce some nervous manifestation or general arterio-sclerosis, however vigorous the treatment may now be, it is impossible to convert a positive Wassermann's reaction into a negative one. Therefore, treatment at this stage can never cure the patient, only arrest the further progress of the disease, and, as the lesions are due to the syphilitic virus, it is sufficient indication to give mercury and not rely solely on potassium iodide, as is so frequently done. The only exception should be in general paralysis of the insane and tabes, conditions which are not infrequently aggravated by the use of mercury.

Arsenical Treatment.—Anæmic and tuberculous folk who subsequently contract syphilis are often unable to stand mercury. Mercury makes them weak and more anæmic, when resource should be had to arsenic, which is a valuable drug in any stage of the disease. Atoxyl and the other aryl-arsenates have been vaunted as ideal preparations, but I have witnessed two cases of temporary blindness, and one of persistent headache following their use. They are, therefore, not to be recommended to any practitioner who has to live upon his reputation. It must also be remembered that these organic arsenical preparations so affect the system that a repetition of their use some months later may give rise to most alarming symptoms, owing to the patient having been made over-sensitive to their action—a condition analogous to the serum disease. As one can test the over-sensitiveness to tuberculin by means of the Pirquet reaction, the same test can be applied to drugs, and by this means one could pick out those who gave a cutaneous reaction with the aryl-arsenates, as being subjects who would show an idiosyncrasy to the drug. A new drug, called Ehrlich-Hata's preparation, No. 606, which is dichlorhydrate, dioxdiamido-arsenobenzol, has just appeared, which is said to work a most wonderful change in syphilitic lesions at any stage of the disease after one injection of 5 gr. Hata has already used it in Frankfurt in more than 100 cases of syphilis without meeting with any untoward symptoms. Out of 25 patients reacting positively, 23 gave a negative Wassermann after one injection.

Syphilis in Mother and Child.—Owing to the fact that a woman who aborts or bears syphilitic children is herself immune, because she is syphilitic, she should be treated whether she shows symptoms or not, throughout the whole time of each successive pregnancy, with the hope of begetting a normal child. It is important also for the father to receive treatment, should he give a positive Wassermann's reaction. A child born of a suspected syphilitic mother should, if a positive Wassermann be obtained, be treated for at least two or three years. No hard-and-fast line can be drawn as to how long treatment in such cases is necessary, owing to the fact that congenital syphilitics give a positive Wassermann, whether under the influence of mercury or not. Too much stress cannot be laid upon the importance of testing a woman's blood who aborts or has premature labours. In drawing up the statistics on infant mortality it will be noticed how small a rôle syphilis seems to play, for the simple reason that more congenital syphilitics are born dead than alive. Hence it is so important to get every syphilitic woman under treatment as soon as possible. A child

born of syphilitic parents may show no signs until some time after birth; therefore, all children born to suspected parents should, whether they show symptoms or not, if they give a positive Wassermann, be treated with mercury, since by so doing we shall be able to avoid the horrors of juvenile tabes, double interstitial keratitis, &c. Hochsinger since 1869 has been able to keep under supervision 134 women who showed no signs of syphilis but begot syphilitic children. These women gave birth 569 times, out of which 253 were born dead, 44.4 per cent.; 263 were syphilitic, and 53 were without a taint; of the 263, 55 died before the fourth year—over 20 per cent. That treatment of the parents has an enormous influence on the severity of the disease in the offspring is shown so well in private cases, where the parents are more systematically treated and the number of syphilitic children are fewer and the disease less severe.

Surgical Section.

June 14, 1910.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

Discussion on the Present Position and Treatment of Syphilis.¹

Mr. C. F. MARSHALL, in reopening the discussion, said: It appears to me that the present position is this: the treatment of syphilis is drifting too much into the hands of manufacturing chemists. We are asked to abandon the simpler preparations which have stood the test of time in favour of new-fangled and complicated preparations, too often of unstable composition. One new preparation is followed by another with such frequency that it is impossible to test their value. This applies especially to new forms of mercurial injections and new arsenical preparations. With regard to injections, I think the method of injection is more suited to acute than to chronic conditions—that is, to conditions where rapid action is required. Hence, mercurial injections should be employed where the rapid action of mercury is indicated. However, I prefer *inunction* in such cases. Injections are no doubt useful in Army and Navy cases, but irksome and too expensive in private practice. They are not suitable for the routine treatment of syphilis. All ordinary cases can be treated by ingestion. With regard to aryl-arsenates I consider them not only unnecessary but absolutely contra-indicated, on account of the possibility of *blindness* resulting, many cases of this

¹ Adjourned from June 7.

sequel having been reported. Syphilis by itself is quite bad enough without the addition of blindness. The multiplication of new drugs is possibly due to insufficient knowledge as to the use of old ones. Iodide of potassium is useful in all stages of syphilis—not only in tertiary syphilis, as was formerly taught. With regard to excision of the chancre, the evidence is contradictory. The earliest possible excision has often failed, as has also excision of the point of inoculation before the chancre has appeared. On the other hand, Neisser's experiments seem to show that excision is worth trying; but in most cases it involves too much mutilation. To be successful the spirochaetes must be localized to the chancre; their spread into the general circulation may result from unskilful excision.

Dr. GEORGE PERNET said that in this country and in the United States the profession was behind the times in regard to methods. He was surprised that there should be such a small attendance as the present one when such an important disease as syphilis was under discussion. Some French observers were of opinion that at least one-third of the morbid conditions met with in the practice of medicine were due to syphilis. John Hunter, whose great authority put people off the scent with regard to syphilis and gonorrhoea, especially his opinion that syphilis never affected the viscera, was known to have been quite wrong in his views. Another great authority—Ricord—had maintained that secondary lesions were not infectious. Therefore it was important in this discussion to disregard mere authority. The great point was to investigate with an open mind.

There were now ample means of diagnosing syphilis, and he need not enumerate them. He did not think, however, that examination of the cerebrospinal fluid had been sufficiently insisted upon. There was now little excuse for leaving a case to run on until (so-called) secondary symptoms appeared. He had seen many cases in which four or five weeks had been lost, and even that had made a great difference in the complications which followed, especially in the case of extra-genital infections. He considered the pill treatment had failed; otherwise why should our asylums contain so many patients suffering from general paralysis of the insane, and the nerve hospitals be so busy with tabes, to mention only two of many diseases? Sometimes circumstances compelled the giving of pills, but he did not consider that a good form

of treatment. Our present attitude, which led to the ignoring of syphilis, was bad both from the national and the private points of view. Dr. Mauriac, of Bordeaux, had recently presented a thesis before the University of Bordeaux¹ comparing the effects of syphilis treated by pills and potions on the one hand, and on the other by intramuscular injections, either soluble or insoluble preparations. He showed that cases treated by pills gave a positive Wassermann in 67 per cent., but in those treated by intramuscular injections only in 22 per cent. Rohde had published a paper in the *Dermatologische Zeitschrift*² in which he gave details of comparative experiments based on the Wassermann test with patients treated by calomel, inunctions, and salicylate of mercury. Calomel came out first, inunctions second, and salicylate of mercury a long way behind. Dr. Pernet agreed that inunctions were very useful, but they had disadvantages and were less accurate than injections. In civil private practice it was difficult to get some patients to accept inunctions. The two insoluble preparations he used were calomel and grey oil—both very valuable. Calomel came first, and was a sovereign sheet-anchor; but he reserved it, as a rule, for bad cases: for instance, where there was threatening cerebral trouble, optic neuritis, and so forth. Calomel was also most valuable in the obstinate palmar syphilides and in some tongue cases. Every syphilitic patient should be dealt with on his merits; the urine should be examined, and everything should be done to adapt the treatment to the particular case. He used various preparations, both soluble and insoluble. The decision which to use was the result of experience, and depended on various factors. Sometimes intramuscular injections of insoluble preparations were contra-indicated. Recently he (Dr. Pernet) had had a case under his care in which, five to six months after contracting the primary sore, optic neuritis supervened. He started the patient at once on 10 cg. of calomel, and followed this up. The calomel injections had done good. If such a patient were treated with pills he would expect the optic neuritis to get worse and go on to optic atrophy. Dr. Pernet admitted that calomel treatment was sometimes painful, and he always warned patients as to this, pointing out at the same time that it was worth while putting up with some pain in order to avoid serious com-

¹ Mauriac, "Le Séro-diagnostic de la Syphilis, 1909."

² Rohde, "Welche Quecksilberkur ist die beste?" u.s.w., *Derm. Zeitschr.*, Berl., 1909, xvi, p. 349.

plications. The French calomel preparation, containing 40 cg. to 1 c.c., was painless in some cases, but not in others. On the other hand, Zambelletti's calomel gave very little pain as a rule. As to grey oil, that could be used in a number of suitable cases. Grey oil, 40 cg. to the cubic centimetre, was now in the French Pharmacopœia.

Notwithstanding ideas expressed about the scientific chemists, Dr. Pernet considered medical men were much indebted to them for what they had done in connexion with preparations for syphilis.

It was important to use a needle sufficiently long. He used a 5 cm. needle of iridized platinum, and even one of 7 or 8 cm. long in very fat patients, for it was necessary to get into the muscle; it was no use putting a needle only 1 in. long into the buttocks, when using insoluble preparations especially. There were drawbacks to the insoluble preparations. He employed them in selected cases and for serious threatening symptoms.

Treatment should be carried on for at least four years, and in the following way: For two years there should be vigorous treatment, and for two years the patient should be treated less vigorously, unless symptoms necessitated stronger measures. After that patients should have further courses at intervals. Fournier had found, from an analysis of his cases of general paralysis, that 65 per cent. occurred between six and twelve years after contracting the chancre, and he therefore advised another course of vigorous treatment in the seventh or eighth years. Dr. Pernet thought that was rational, and he considered it good practice, especially if there were a bad nervous family history.

It was important that the patient's teeth should be put in order. He seldom found the mouth very bad in private patients, but a dental surgeon should attend to the mouth before commencing injections, or indeed any mercurial treatment. Sometimes it was well to test the patient with a soluble preparation before using an insoluble one. The soluble preparations were more quickly eliminated than the insoluble. In addition to benzoate of mercury (Professor Gaucher's hypertonic formula is valuable), the sozoiodolate of mercury and many other preparations are useful. In the treatment of early syphilis iodides were not necessary as a routine practice, at least in patients under 40 years of age, but at that age and over iodides should be employed on account of arterial changes. Iodipin and lipiodol were very useful, especially in intramuscular injections in bad cases which

resisted ordinary iodide treatment. Iodides were, of course, valuable in various conditions, either alone or in combination with mercurial treatment.

As to technique, he had so recently given details in a paper read before the American Medical Association at Atlantic City last year—that it was not necessary to go into that.¹

With regard to atoxyl and its derivatives, it had now been settled by thorough investigation at the St. Louis Hospital that treatment by atoxyl had failed, though it was useful as an adjunct in some cases. He referred again to a case which developed optic neuritis five to six months after the original chancre, mercury by mouth only having been given until he saw the case. If that patient had had aryl-arsenates, or soamin, or atoxyl, the atoxyl might have been blamed for the optic neuritis. Howbeit, these organic arsenical bodies were not innocuous, and makers should exercise care in their advertisements. He had found that when it was necessary to stop the mercury, quinine and salicin were helpful. Mercury was the only fundamental treatment of syphilis.

Extragenital chancres were frequently overlooked, and students should be taught to diagnose them more than they were. Students often ran great risks of infection in early years of hospital work—when attending midwifery cases, for instance, without the necessary knowledge of syphilis. An important point was the treatment of cases of mixed infections—that is, by the *Treponema pallidum* plus some other micro-organism. For these he believed vaccine treatment had a place. An attempt should be made to identify the other organism, in order to prepare a vaccine if possible. He insisted on the importance of the proper teaching of syphilis. The great pandemic prevalence and the serious results of syphilis entitle it to a prominent place in the curriculum. On the Continent men were better trained to understand and treat syphilis and its protean symptoms than was the case with us. In several centres abroad systematic teaching obtained, and in some Universities lectures on the subject were delivered to the students of all the faculties, including that of theology.

With regard to the treatment of pregnant syphilitic women, treatment did not always result in a living child. In such cases intramuscular injections of soluble salts were to be preferred to insoluble

¹ Pernet, "The Intramuscular Treatment of Syphilis, with especial reference to the Insoluble Preparations of Mercury," *Lancet*, 1909, ii, p. 212.

preparations. With regard to congenitally syphilitic women, they were very liable to repeated abortions. Dr. Pernet had published a very significant case of the kind.¹ These women required treatment during pregnancy.

As to the question of special Acts, it is one that should be taken on its merits, according to geographical latitude, race, and degree of civilization of the people concerned. In Russia, where there was official inspection, the system had practically failed, and at the present time there was a movement in France in opposition to the official methods.

Mr. H. W. BAYLY: Anything that I can say on the important subject of syphilis and its treatment must necessarily be from the point of view of the pathologist, and I suppose that all will agree that it is in the pathological rather than the surgical or purely clinical aspects that the greatest advance has been made in the last six years, and it is to the pathologist that the clinician or surgeon now turns for diagnosis in difficult cases. By the help of the reflecting immersion-condenser, commonly called the ultra-microscope, which I had the pleasure of demonstrating before the Clinical Section of this Society in November last, the *Treponema pallidum* can be easily detected in scrapings from the site of the inoculation, even before any definite chancre has developed. Much valuable time is thus gained, and the patient is saved the discomfort of the secondary manifestations. I cannot agree with Dr. Fleming's opinion in regard to the value of the Chinese-ink method, as it is frequently very difficult to differentiate the various spirochætes by this method, and the diagnostic value of the movements is lost. Captain Harrison agrees with me that this method is far inferior to dark-ground illumination.

Neisser has shown, by means of the Wassermann reaction, that the earlier that anti-syphilitic treatment is commenced the more probability there is that early and permanent cure will result, and therefore the ultra-microscope, which, in the absence of local antiseptics, settles at once whether the commencing pimple or ulcer is the site of a syphilitic inoculation or not, must be considered of the greatest possible value, both from the point of view of surgeon and patient. By the help of the ultra-microscope the treponema can also be detected in scrapings of a

¹ Pernet, "The Offspring of Congenital Syphilitics," *Brit. Journ. Dermat.*, 1899, xi, p. 459.

papule or mucous plaque, but it is on the phenomenon of the development of a substance in the serum of syphilitics that has the property of combining with another substance present in organ extracts, and in this process of combination using up complement, that we chiefly rely for the diagnosis of late or latent syphilis. This complement-fixing test, first worked out by Professor Wassermann, and which carries his name, requires a menagerie of sheep, guinea-pigs, and rabbits, as well as the liver of a syphilitic foetus before the original technique can be undertaken. It has been considered by some devotees that the oracle demands too many sacrifices before she will speak and give her verdict, and numerous workers have tried to simplify the test. But it seems to me that the most essential of all factors in a *comparative* test such as this is that all factors, except the one to be tested, should be *constant*.

In the technique that I use and advocate I mix complement and syphilitic antigen and normal saline solution in bulk, and measure off 2 c.c. to 5 c.c. of this mixture into each test-tube. The only variant that I use is the serum to be tested, of which I add 0.3 c.c. to each tube. In the same way I mix the hæmolytic antigen and antibody in bulk. Any difference in the amount of hæmolysis between the tubes must therefore be due to the only variant factor—viz., the serum tested.

With Hecht's or Bouer's method, however, or the modification of these methods advocated by Dr. Fleming, the only constants we have are the syphilitic and hæmolytic antigens. The complement and hæmolytic antibody, as well as the syphilitic antibody, are all variants. Some human sera contains no hæmolytic antibody to sheep's corpuscles; others contain some, but in variable quantities. The complement-content of human serum also varies greatly. Papers by Dr. Fleming and myself, giving our different techniques, were published in the *Lancet* of May 9, 1909.

I note with great interest Captain Harrison's remark that the patient's serum is completely deplementized by heating at 55° C. for ten minutes, and that greater delicacy of reaction is obtained than if the serum is heated for half an hour. Benefiting by the advice and experience of Captain Harrison, I now invariably use two strengths of complement, and can thus obtain a roughly quantitative measurement of the complement-fixing power of the serum. Frequently in this way we can obtain an indication that the patient is getting under the

influence of treatment, and some hæmolysis will occur in the tube containing the larger dose of complement, although the smaller dose of complement is completely fixed. By this means I have investigated the therapeutic value of the different anti-syphilitic remedies, and I published with Mr. Charles Gibbs, in the *Lancet* of May 7 last, a preliminary note of the result obtained in 231 cases which had received various methods of treatment, extending over various lengths of time. This was, I think, the first communication on this subject made in this country.

I was much interested to hear from Captain Harrison last week that he also has found that pill treatment has a very slow action on the Wassermann reaction compared with intramuscular injection. My work on this subject has shown that six months' pill treatment has no effect on the Wassermann reaction, and that there is no trace of hæmolysis even with the double dose of complement. On the other hand, 82 per cent. of cases treated by inunction and 35 per cent. of cases treated by injection showed some effect of treatment after only three months. As far as I have gone, therefore, my conclusion is that inunction is the best form of administration of mercury, and I gather that Mr. McDonagh's large Continental experience leads him also to this opinion. Calomel injection seems much better than pill treatment, but not so good as inunction, as estimated by the Wassermann reaction. Potassium iodide and the aryl-arsenates have no effect on the Wassermann reaction, as far as three months' treatment is concerned.

If the technique advocated by Dr. Fleming is used, besides having the grave disadvantage of replacing constants with variants, any quantitative measurement is obviously out of the question, as if he depends on the patient's serum for complement and hæmolytic amboceptor, as well as for syphilitic antibody, he obviously cannot increase or diminish one factor independently of the others. I have tried Dr. Fleming's technique, and the technique that I use, side by side in 200 cases, and have come to the conclusion that in the long run Dr. Fleming's technique does not save time or trouble, and is not so reliable as Wassermann's original technique as modified by me. I have found partial or complete absence of hæmolytic amboceptor for sheep's corpuscles in 10 per cent. of human sera; and even if the amount of complement were constant, which it is not, these 10 per cent. would have to be done again by another technique than Dr. Fleming's, and the time spent on the first useless test would have been entirely wasted. I agree with Captain Harrison that the

cases most likely to give fallacious results with Dr. Fleming's technique are those intermediate cases where only a *small* amount of complement or hæmolytic amboceptor is present. Most authorities also consider that complement in fluid serum deteriorates quickly, and that it is unsafe to use complement more than twenty-four hours old. Yet another source of error, therefore, is admitted if Dr. Fleming's technique is used with sera more than twenty-four hours old. I have therefore ceased using Dr. Fleming's technique.

In a paper read by Dr. Dean before this Society and in the discussion following it, the author of the paper, Dr. Henderson Smith, and Mr. McDonagh, spoke in favour of the original technique as compared with the so-called simplified methods, and I am interested to hear that Captain Harrison's large experience has led him to the same conclusion. I think it is evident that there is hardly any English pathologist who has had any large experience of the Wassermann reaction, with the exception of Dr. Fleming, who is in favour of any simplified method.

It is in the pathology of syphilis that the recent great advance in our knowledge of this disease has been made, and I think that now the surgeon cannot, as in the days of old, claim this disease as entirely his. Why a chronic spirochætosis should be considered surgical at all is baffling to the mere pathologist. And yet not long since I heard my eminent colleague who opened this discussion refer to the pathologist as an "accessory" to the surgeon. On the contrary, I think that perhaps the day may not be far distant when the surgeon will be looked on as the rather dreaded accessory of the physician-immunizator-pathologist, and that his help will chiefly be sought for surgical interference where his skill in operative technique will render his help essential. In syphilis it is by the aid of the microscope that we learn whether a suspected sore is syphilitic or not; the pathologist alone can diagnose latent syphilis, and he alone can venture an opinion as to whether the patient is free from the disease. The pathologist can say when treatment should be begun, whether it is effective, whether syphilis is the cause of repeated miscarriages, whether the tumour or rash is syphilitic or not, when the patient may marry, &c.; and it is only in clear cases that the surgeon can diagnose this disease unaided by the pathologist. I claim, therefore, that at any rate in regard to syphilis the pathologist is equal and more than an *accessory* to the surgeon. The surgeon, gynæcologist, or dermatologist certainly *treats* the disease, but

nowadays his treatment is guided by the light of pathology as well as by that indispensable clinical experience which the clinician possesses and which the pathologist does not. If the future holds a serum treatment for syphilis, as does not appear impossible, will it be possible then to still rank this disease under the surgical banner? Why should infection with the *Treponema pallidum* be surgical while infection with the trypanosome or filaria is medical? Surely all three are a mixture of the two, and generally far more medical than surgical. Unless the microscope, the test-tube, or the guinea-pig, rather than the knife, is in future to be the emblem of the surgeon, it is difficult to imagine that syphilis can remain for very much longer a surgical disease, at any rate to any greater extent than tuberculosis can be considered a surgical disease.

In conclusion, I should like to emphasize my opinion that anti-syphilitic treatment should not be delayed till secondary symptoms have developed, but that a scraping of every doubtful primary sore should be made and treatment begun at once if the *Treponema pallidum* is found. I consider also that the Wassermann reaction should be used as a guide to treatment, which should be continued intermittently until a series of negative reactions is obtained after all treatment has been left off. I consider that oral treatment is the least satisfactory. I am entirely in accord with Captain Harrison's opinion that it is high time that legislative steps should be taken to prevent the spread of syphilis. We live in the days of preventive medicine, and it speaks ill for the courage and dignity of our profession that, through fear of the anger of Exeter Hall and faddist politicians, we have allowed these unpractical extremists to dictate to us on questions of public health. Surely a united medical profession could insist on syphilis being ranked as a notifiable disease, and on the segregation of infectious prostitutes. By this I do not mean that I am in favour of replacing the old Contagious Diseases Act on the Statute-book, for I am against the licensed-house system, but I should like to see syphilis treated like scarlet fever or smallpox, with efficient regulations and penalties to insure against the spread of the disease.

Mr. JONATHAN HUTCHINSON said that so much good work had been done in the investigation of syphilis by surgeons and pathologists in this country in the past that he could not help expressing a regret, which all must feel, that the two recent great discoveries had not been due to English pathologists: he meant the discovery of the spirochæte

and Wassermann's reaction. Wassermann's reaction was so complex, and depended on so many unprovable hypotheses, that it was marvellous anyone should have thought of it; but the discovery of the spirochæte ought not to have baffled so many English and other workers. Now that it had once been made, all our pathologists remarked how easy it was. He agreed it was pathologists who must speak with authority about the Wassermann reaction. He gathered that it became negative for a time after intramuscular injections of mercury had been given, but that after a time it again became positive. He urged that was a strong argument in favour, not of interrupted courses, but of continuous treatment for at least two years. In cases of inherited syphilis, he gathered that the Wassermann reaction was present whatever courses of intramuscular injection were given. That was a most interesting statement to follow up, and he reserved his belief until others had tested the point. If it was true, it was a commentary on the advantages of intramuscular injections. In Mr. Lane's admirable review it was urged that interrupted courses of mercurial injections probably afforded the best treatment, and that that should be carried out for four or five years. The fault of the treatment at Aix was that patients had a course of injections, or inunction, but were told nothing as to continuing with the mercury, though they might be told to come back in six months for another course. He had seen that system fail to prevent speedy recurrence of secondary symptoms. As to the clinical results of keeping the patients under the continuous influence of mercury for eighteen months or two years, with due precautions as to abstention from alcohol, it would be wearying to the meeting to go into detail. He was confident that nine out of ten patients treated continuously by oral administration in that manner remained free from symptoms for many years afterwards, as long as one could follow them up. Many of them married and had healthy children. Not a few patients who had been treated by oral administration of mercury, after some years, some as short a time as eighteen months from their first attack, contracted a primary chancre and the symptoms of syphilis all over again. He had narrated a series of such cases at the Section last year, when he drew attention also to thirty cases of the kind recorded by his father, and adduced them as strong evidence in favour of the belief that oral treatment was often completely efficacious, the patient being so cured that he contracted the disease again.

There were not many points in the discussion on which unanimity had been approached, but all were agreed that the much-vaunted arsenical preparations were not only of little use, but were very dangerous. When a well-known firm published the statement that soamin and the other preparations possessed great advantages over treatment by mercury, and that they had slight toxic action compared with inorganic preparations of mercury, he took the strong step of writing and obtaining an interview with them, telling them there was no foundation for the statement and that they were running great risks in making it. We all knew the remarkable predilection of arsenic for the nervous system, and the practitioners who used it in large doses ran great risks. Perhaps partly due to that interview, and partly to the bad results on the optic nerve, the firm had now issued a warning stating that arsenic was not to be injected in the treatment of syphilis "if there is any tendency in the patient to degenerative changes in the retina or optic nerve." Everybody degenerated sooner or later, and he would ask who could detect the early signs of it. The second condition was "the absence of any disease of arteries, kidneys, or liver," a very difficult thing to establish. The firm said, thirdly, "the visual fields should be tested at regular intervals, as eye symptoms might come on without preliminary poisoning symptoms, such as vomiting or gastric pain." But the carrying out of routine testing of the visual fields would not only be beyond the range of physicians and surgeons unless they had studied ophthalmology, but would naturally alarm the patient. Moreover, he could not understand the practitioner using arsenical preparations when he remembered that retrobulbar neuritis, once started, tended steadily to progress. Another result of the discussion, he thought, had been to consign to oblivion the treatment by injection of soluble preparations of mercury, as they were not only risky, but had no advantages.

There remained, finally, the old controversy regarding intramuscular injections and treatment by the mouth. He granted that the evidence as shown by the Wassermann test seemed strong. Mr. McDonagh said the intra-oral treatment was doomed, but the evidence from other sources hardly bore out Mr. McDonagh's strong prediction. Threatened men and threatened causes often lived a long time. It might be found that the best treatment would be a course of mercurial injections, followed by a long-continued administration of mercury by the mouth. His personal experience had been as follows: The great majority of

cases, granted they were seen early and treated as soon as diagnosed, could be treated satisfactorily with a complete absence of symptoms. But it was admitted that some cases did not respond as it was hoped they would, and the question arose—Were intramuscular injections more successful? His experience of them in such cases had been, on the whole, disappointing, and that was especially true in the troublesome cases of relapsing ulcers of the tongue and throat. In one case of this kind the patient had been through no fewer than twelve courses at Aix, and when seen afterwards he was not only no better, but even worse. He agreed that many cases of superficial glossitis due to an old syphilitic taint were made worse by mercury, given in any form. Dr. Lieven had urged the profession in England to give up old-fashioned ideas and employ mercurial injections universally. But that gentleman's paper showed that he had seen so many complications from intramuscular injections that he had discarded them in favour of inunction. Major French's evidence in the discussion had been very interesting—namely, that he had seen more persistent symptoms with intramuscular injection than with either inunction or intra-oral administration. He had asked as many Army surgeons as he could on the point, for their experience was larger than that of civilian surgeons, and many of them had told him that they had not been satisfied with intramuscular injections, and had seen better results from mouth treatment. It was rather hitting below the belt to say that the worst cases of tertiary syphilis were seen in those treated by a long course of grey powder. It would be equally truthful to assert that many bad cases of tertiary syphilis had been treated throughout by inunction or injection.

But how far was the discussion advanced? Not at all. All the speakers had stated that mercury was the sheet-anchor for treatment, and that iodides were only useful in the tertiary stage. But in the secondary stage one sometimes saw very troublesome ulceration of the tonsils, which resisted mercury, however given and however pushed. One recognized them as amenable to sodium iodide and the liquor, or the iodide alone. He would be glad if Mr. Lane would state in his reply what was the practice now at the Lock Hospital. At the time the speaker was on the staff there only the minority received inunctions or injections, and he was interested to know whether the majority received injections now. It was a test question—Which was found to be the most practicable and efficient treatment in the wards and in the out-patient department?

One interesting point which had emerged from the discussion was that one must keep one's patient under mercurial treatment for at least two years, and Mr. Lane had urged that the injections should be continued for two or three years more. In this he had the support of many Continental authorities. One might hope in the future to obtain a more rapid, safe, and pleasant cure of the disease, but at present it must be insisted on that mercury long continued was the right treatment. As to the particular method by which it was administered, he urged that, so far as temperament would allow, we should keep an open mind and avoid "the falsehood of extremes."

MR. ERNEST LANE, in reply, said the criticism of his opening remarks had been so favourable that but little was left for him to respond to. The general result had been to emphasize the value of the recent pathological discoveries—the spirochæte and the Wassermann test. He thought most of those present would not go so far as Mr. Bayly and hand over the treatment of the disease entirely to the pathologist. He (Mr. Lane) considered the pathologist as an accessory to the surgeon, and a very valuable one; but he would not like the pathologist to deprive the surgeon of one of his means of livelihood. Condemnation of the use of the arsenical preparations for syphilis appeared to have been fairly unanimous. It was a pity one of its principal advocates had not been present at the discussion, as one might then have heard a word in its favour. Mr. Hutchinson's remarks as to his interview with a representative of the firm were of interest to him, as he was himself having interviews with the same man presumably, and he had informed the representative of the occurrence of a case of blindness in his own clinic from the drug. Still, the favourable notices concerning soamin and orsudan continued to appear in the firm's books. Early commencement of the treatment and its longer duration had been well emphasized. He believed the treatment should be continued for five years, and for neurotic patients even seven years. He did not agree with Mr. Hutchinson that there should be continuous treatment for two years—a patient could not be kept under mercury for that time. In future, he thought, the treatment should be regulated by the pathologist. He hoped surgeons would continue to treat patients, but that the pathologists would help them by giving the results of the blood tests.

Dr. Pernet had condemned the pill method in a rather sweeping fashion, but he was not sure that he had sufficient grounds for doing so. The majority of cases in the last fifty or sixty years had done fairly well under pill treatment, as evidenced by the fact that syphilis was now a very mild disease. The presence of large numbers of patients suffering from the remote effects of syphilis in our asylums had been mentioned; but he did not think cases were in asylums because of the pill treatment, as suggested, but because they had been insufficiently treated by pills, a method which he did not think should be cast aside in the way which had been advocated. He had pointed out in many papers that one of the principal factors in prophylaxis was education on the subject, and he was at one with Dr. Pernet that there was great neglect on that matter in this country. There were very few clinical remarks made on syphilitic cases in our general hospitals; if more instruction were given on that subject, he believed the disease would be even milder and less prevalent than at present.

In answer to Mr. Hutchinson, the routine treatment he adopted at the Lock Hospital was calomel injections, for the reason he had given: that the patients were thereby much sooner got under the influence of mercury, their symptoms more rapidly disappeared, and they were able to return to their avocations at an earlier date than if other plans had been adopted. They were advised to present themselves subsequently at the hospital for less drastic treatment. He believed most of his colleagues in charge of in-patients used intramuscular injections entirely, and mostly of insoluble salts. It was not easy to give intramuscular injections in the out-patient department; it was difficult to ensure asepsis, and there was danger of abscesses. He was in opposition to the Contagious Diseases Acts as a means of prophylaxis for syphilis. He had been connected with the Lock Hospital thirty years, partly at the time when those Acts were enforced, and for a long time since their repeal, and he agreed with most Continental surgeons that the Contagious Diseases Acts were of very little use indeed. Syphilis was as prevalent in Paris as it was in London, and it was due to the fact that the Acts could be evaded with ease. The amount of clandestine prostitution in Paris was enormous, but the number of prostitutes on the register was comparatively small. The disease was disseminated not by those on the register, but by those not registered as prostitutes. But the subject was too vast to deal with at such a meeting as this. Mr. Hutchinson said he (Mr. Lane) had regretted the advocacy of intravenous injection,

but he did not. He had had considerable success from the use of cyanide of mercury as intravenous injections, and he would continue to advocate it if he were not in possession of a more valuable method—namely, intramuscular injection as an insoluble salt. Little had been said about iodides during the discussion, but Mr. Hutchinson had called attention to their value in certain cases; on the syphilitic virus, however, they had practically no effect. Certainly in all cases of syphilitic ulceration of the tongue, lips, and fauces one had in iodides, possibly combined with mercury, a most valuable remedy.

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VOLUME THE THIRD

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1909-10

THERAPEUTICAL & PHARMACOLOGICAL SECTION



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1910

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THERAPEUTICAL AND PHARMACOLOGICAL SECTION.

CONTENTS.

October 5, 1909.

	PAGE
A Discussion on the Teaching of Therapeutics in the Hospital Wards. Opened by Sir T. CLIFFORD ALLBUTT, K.C.B., M.D., F.R.S.	1
<p style="margin-left: 40px;">Professor OSLER, F.R.S. (p. 7)—Dr. SAINSBURY (p. 9)—Dr. CALVERT (p. 10)—Dr. R. HUTCHISON (p. 11)—Professor DIXON (p. 12)—Dr. BEDDARD (p. 14)—Sir DYCE DUCKWORTH (p. 14)—Dr. HACKNEY (p. 15)—Professor CUSHNY, F.R.S. (p. 15).</p>	

November 2, 1909.

The Physical Signs of Incipient Pulmonary Tuberculosis and its Treatment by Continuous Antiseptic Inhalations, with the Results in Thirty Cases. By DAVID B. LEES, M.D.	17
The Action of some Diuretics. By H. H. DALE, M.D., and P. P. LAIDLAW, B.C.	38

December 7, 1909.

A Discussion on "The Therapeutical Value of the Lactic-acid Bacillus." Opened by GEORGE HERSHELL, M.D.	51
<p style="margin-left: 40px;">Dr. GOODBODY (p. 56)—Dr. VAUGHAN HARLEY (p. 57)—Dr. BULLOCH (p. 58)—Dr. EYRE (p. 59)—Dr. R. HUTCHISON (p. 60)—Dr. MILLER (p. 61)—Dr. HERTZ (p. 62)—Dr. LANGDON BROWN (p. 62)—Dr. HERSHELL (p. 62).</p>	
A Fatal Case of Poisoning by Phosphorus, taken as an Abortifacient, with Unusual Subcutaneous Hæmorrhages. By REGINALD G. HANN and R. A. VEALE, M.B.	64
Report on a Drug known as "Kangalugi." By H. WIPPELL GADD	67

February 1, 1910.

	PAGE
The Immunity Reaction to Cancer. By E. F. BASHFORD, M.D.	69
Proprietary, Patent, and Secret Remedies. A Discussion opened by W. E. DIXON, M.D.	82

March 1, 1910.

Discussion on Proprietary and Secret Remedies (*continued*).

Mr. T. P. BEDDOES (p. 91)—Mr. F. W. GAMBLE (p. 93)—Dr. J. GRAY DUNCANSON (p. 94)—Dr. W. MARTIN (p. 95)—Mr. W. J. UGLOW WOOLCOCK (p. 96)—Mr. E. F. HARRISON (p. 97)—Professor CUSHNY (p. 99)—Professor DIXON (p. 99).

The Treatment of Infective Endocarditis. By JOHN COWAN, M.D.	101
---	-----

May 3, 1910.

Some Points in the Treatment of Gastric Ulcer. By CHARLES BOLTON, M.D.	117
A Criticism of some Principles in the Treatment of Chronic Nephritis. By W. LANGDON BROWN, M.D.	138

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Therapeutical and Pharmacological Section.

October 5, 1909.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

A Discussion on the Teaching of Therapeutics in the Hospital Wards.

Opened by Sir T. CLIFFORD ALLBUTT, K.C.B., M.D., F.R.S.

SIR CLIFFORD ALLBUTT said that as he had been assured that no formal paper would be required in the discussion, he had less hesitation in accepting the honour of opening it. And even before the President, a pharmacologist whose attention is concentrated upon a more or less abstract form of thinking about therapeutics, he would take up, without hesitation, the position which indeed was that proposed to-day—namely, that of the practical man in medicine. For he had ample reason to know that the pharmacologist, on the one hand, was full of sympathy with the purposes of the physician; while the physician, for his part, saw as clearly the incalculable gain which was to come to him sooner or later—in some instances, indeed, immediately—from the investigations of the laboratory. But the question in the present discussion was, What was to be done in regard to therapeutics at the bedside? What example were we to set at the bedside before the student? It was good to know; but their business was to do, and doing was the main purpose of the physician. People looked to the physician to produce results. Laboratories were necessary; science was essential; but therapeutics was not a science, or not science only, but an applied art; and it might be a long time before, in this or that sphere of pharmacology, science could catch them up and come very definitely to their assistance. Until that distant day arrived they had, indeed, to distrust working on

principles; to beware even of certain of the perils of acting on principle. Medical practice is too much embarrassed by the incessant contingencies of clinical experience to be able to take a ruler and rule out a course of action on broad lines of principle. They might rather be looked upon as pioneers, traders, and adventurers, going into an almost unknown—or, at any rate, an unsurveyed and uncharted—country, and, for the purposes of their adventure and trade, making their way through the wilds as well as they could. In the course of tens, twenties, or hundreds of years the surveyor would lay down the various features of the region, and lay out its roads with exactitude; but it would have been a poor thing for geography if adventurers had waited for the cartographers. Therefore he thought, as concerns the present question, the Section must for the moment dissociate itself from pharmacology in the sense of disinterested science; and, moreover, he thought it very important that the difference of these two points of view should be explained very clearly to the student.

If the students who went into Professor Cushny's or Professor Dixon's or other pharmacological laboratory thought they were going to obtain therefrom practical maxims, rules which would guide them in their work as artists and physicians, he believed they would find themselves disappointed. It would not be a fruitful way of developing their clinical resourcefulness to lead them to expect immediate practical results from the researches carried on in the laboratory, away from the bedside. But he thought we ought to point out to the student how the matter is approachable from these opposite sides; how, by sheer empiricism or by partial empiricism, the practical man, having tracked out rude paths of his own, would probably find them in their main directions verified, although probably very much corrected by the pharmacologist when he caught the practical man up. But the practical man—the artist—must always be, as he always has been, very considerably in advance of the researcher, the scientist; he must be doing, and in the stress of action there can be no waiting for the corps of surveyors and map-makers. Therefore, therapeutics must be approached from opposite sides; the student must be taught that rule of thumb comes first, and that verifications follow very slowly behind. The pharmacologist had thrown valuable light on practical methods, but, fortunately, most of these methods were known before. For the practical methods were largely a matter of knack. Only a few days ago a shrewd practitioner said to the speaker, "What chaps you turn out from Cambridge! The other day one of them went to see a sick old lady for me, and came away

with his mouth so full of long words, proposals for blood examinations, and so forth, that I was quite alarmed, and went to see the woman, who had been under treatment three or four days with no benefit, and it was obvious at once what was wanted. I gave her a 5-gr. dose of calomel, and the whole difficulty was promptly cleared up. Why could you not teach him simple things like that?" The speaker replied that this half-instinctive rule of thumb was no simple thing, but the fine flower of a lifetime of converse with clinical practice. It was the offspring not of research but of the insight of a shrewd and expert empiricism. That general practitioner would have been unable to explain to Professor Cushny how he had attained to this happy knack, but it was due to the instinctive sense of the artist thoroughly familiar with the materials he had to use, who had gained in time much adroitness in handling them. Now, this was the kind of empiricism which those present would estimate at its full and great value. However, this sagacity was something more than mere empiricism, or mere chance. If these therapeutical methods could not be called scientific—if the President would criticize their vagueness and tentativeness, how they lacked the precision of scientific laws, yet he would recognize also that in practical life we have to act on many rules of that more or less temporary kind which Bacon called *axiomata media*. It had been by means of such rudimentary clinical maxims that physicians have had no little success in their practice; nevertheless, when they could feel the solid bottom of scientific laws it was a rare satisfaction to them. As practical men they had learned meanwhile to manage their many difficulties and contingencies by a sort of instinctive appreciation gained by long watching at the bedside.

Many medical rules and materials came from folk-medicine, a truth which did not seem to be sufficiently realised. He would take three examples out of many from different sides of practice. Digitalis came to us from folk-medicine; and the genius of Fothergill perceived that in this traditional drug—uncertain, tentative, or wild as the use of it may have been—there was no little virtue; and, as a skilled bedside observer, he improved the casual but still empirical routine on which digitalis had been used. Then came the more scientific researches of others, and among the earlier of these he might refer to those of Dr. Milner Fothergill and himself. And in this line of treatment he thought one might almost say that the pharmacologist had caught the clinician up, and had been able so to straighten out and clarify the notions of the practical man that the drug could now be used

with something like precision ; no longer as a kind of blunderbuss.* The second example he would take was vaccination, which again came from folk-medicine. With like genius Jenner saw that there was something in the common rustic notion, and patiently detached the principle from confusing, hampering, contingent circumstances, with consequences so prodigious that one could hardly find an adjective big enough to say what would be the end of its values. The whole structure of immunity now daily rising before us had arisen from that shrewd observation of a tradition of folk-medicine. The third practice of folk-medicine he would cite was poultices. A few years ago medical freethinkers began to scoff at poultices ; and one read of a certain eminent surgeon who was accustomed to say, "Take that dirty thing away and throw it into the fire." Like many of his brethren, the speaker also got rather ashamed of poultices, and the humble practitioner was laughed out of the employment of them, because there was no scientific explanation of their effects. Yet quite recently Bier's method of local congestion had arrived to explain how our fathers did, by proper precautions, compass very considerable benefits by their crude and tentative application of a true principle.

It appeared to him that, if he might venture to touch upon the duty of the pharmacologist, it would be to urge upon him what he knew would be Professor Cushny's own advice—namely, to watch the practitioner, and not to shut himself up in a laboratory in another street. It seemed to him of the utmost importance that disinterested science should be constantly in touch with living realities. Happily, pharmacologists were stepping out of their balloons ; they had learned how valuable were the empirical devices of the doctors, and how these ways of theirs might again and again, as in the cases of digitalis, vaccination, and poultices, just put the pharmacologist on the track and show him where to drive his adits into the mine of truth. But, on the other hand, it was no less the duty of the practitioner to watch the pharmacologist ; because, necessary as were his empirical methods for a time, in their rough application they were at best tentative and wasteful, and not rarely, when misapplied, blundering or even injurious. It was often said to the student that he should not treat "the disease," but should treat the patient ; but this aphorism he could never understand. What could be treated but the patient ? A disease is an abstract idea, and one could not treat an abstraction. This should be explained to the student ; and also that medicine, like politics, is not (or not only) a set of principles, but an art to be exercised, like all arts, in the midst of the shifting

incidents of the particular case. If, then, the practitioner and the pharmacologist would watch each other, a working compromise would be found between the two which would be mutually beneficial. The empiric, oddly enough, needed to be taught how to be an empiric. For unfortunately, he was too often a traitor to his own cause—he would speculate. The empirical schools had done an enormous amount of good in medicine. The great empirical school which sprang up at Alexandria as a reaction against the humoral system of dogmas had been a most valuable discipline for the art of medicine. But unhappily there is no speculator, no visionary, like your practical man. But when one had nothing but empiricism to guide one it must be a genuine empiricism—not be distorted by unchastened speculations. Take, for example, the cure of syphilis—an excellent example of empiricism. We did not know how iodide of potassium or mercury cured syphilis, but the clinical artist had pointed out the way and we could not afford to wander far from it. Before long, no doubt, pharmacologists would be able to say how iodide of potassium cured syphilis, but meanwhile they had to go on the *axioma medium*, the clinical maxim; and they should be careful to tell the student where empiricism begins and ends, and when they should be provisionally content with empiricism, and not involve themselves in speculations and arbitrary hypotheses on matters which would be more properly approached on the accurate methods of the laboratory.

Instead of a genuine empiricism, then, speculations of the most flighty kind had flourished among practitioners, so that in Galen and his endless following there had been a mixture of empiricism with the most unbridled speculations and the most unbridled polypharmacy. Galen cumbered himself with a pile of folk-medicine, much of which had been got together during the early ages, and built up during many centuries of speculative analogies and "sympathies"; and, amongst other things, he inherited the tradition of the celebrated theriac. Galen either thought or pretended to think this antidote so precious that he added thirty more ingredients to it, and declared nobody but himself could make it properly. Even until the end of the eighteenth century this disgusting compound was given, as a matter of routine, to every patient in the Montpelier Hospital. This was not so much empiricism run mad as expatiations of unbridled conjecture. The use of the remedy was not based on observation, however rude, but on the notion that as the viper survived his own poison his flesh would be an antidote to it; and then not only to viper poison, but would be a universal antidote to all poison. Instead of simply watching and record-

ing whether the remedy did good or not, a false empiricism was built on fanciful speculations.

The next point he would urge was that one of the first things to tell the student in the wards was that he was not to be disdainful about therapeutics. As he took up that evening's paper, he had read, "If a man is so clever as not to have any opinions, he is like a nail which is too good to hold down a carpet." If a man was over-sceptical at the bedside he became of less use as a physician. The student should be assured that, apart from scientific laws and pharmacological proof, he was entering into a great inheritance of empirical knowledge of infinite provisional service, so long as it was recognized and used as such; that it was often of crucial efficacy, and would often turn death into life. The student should be assured that such and such have been the facts and results of clinical experience, whether they could be scientifically explained or not. They were to go to the bedside as artists, and thus to do what they could for the patient; if possible on scientific principles, but yet to recognize the tradition of precious empirical knowledge and work which had not as yet received scientific elucidation. Again, one should demonstrate to students the meaning of the Hippocratean maxim of the *vis medicatrix naturæ*. It was part of the extraordinary insight of Hippocrates that he made prognosis one of the great cardinal points of medicine; for prognosis included diagnosis: What was the morbid process going to do? This, again, the scientific pathologist was as yet far from being able to tell us. More and more knowledge was being acquired year after year, yet to explain all our empirical clinical resources and divinations would occupy many a generation to come. In prognosis the family physician would often be able to tell the best consultant in the world whether the patient was going to die or to recover, and he was generally right. Such insight became instinctive, and was born of shrewdness and watchfulness. Therefore, the student should be told not to trust too much to his science schools; he might make his blood-counts, estimate the quantity of albumin found in the urine, cultivate his bacteria, but not forget to look at a case as a whole, and to try to form an idea how the disease was going—an idea only to be formed by watchful practice at the bedside. It had been said that the *vis medicatrix naturæ* might become the *vis devastatrix naturæ*: Nature might do as much harm in one case as she did good in another. But the practitioner learns to appreciate the tendency of all moving systems to recover equilibrium under disturbance—the "righting couple"—and to perceive in the particular case how far this power of righting itself is maintained or endangered, and to adjust and temper his remedial means accordingly.

Finally, it was our duty to warn against the over-use of empirical remedies, seeing that we cannot exactly measure or value them. There had always been a tendency for medical fashion to run to an excessive use of particular means: for instance, of purgatives, or of diaphoretics, or of diuretics, or of venesection, and so forth. He recollected a remarkable passage in the "Timæus," part of which he thought might be used as a motto for the pharmacologist. Plato was animadverting upon the over-use of purgatives. The empirical maxim contained much truth—that of cleansing, or of clearing some foreign matter out of a person: intestinal toxins, for example, in modern language. But it was another thing to over-use purgatives. "If anyone," he says, "regardless of the intention of Nature, would get better of disease and its complications by medicine, by their excessive action he only increases and multiplies them; wherefore we ought always to manage them, if possible, by regimen, such as diet, gymnastics, &c., and not provoke a disagreeable enemy. For diseases should not be irritated by medicines (purgatives, &c.) unless at some critical moment, because they brought other dangers of their own with them." And then followed the reflection which he (Sir Clifford) thought so remarkable: "Since every form of disease is in a manner akin to the nature of the living being (*τῇ τῶν ζῶων φύσει προσέοικε*), whose complex frame has its appointed term of life." Plato might have sat at the feet of Hippocrates, as indeed he probably did. Abernethy said, "I don't like to bully the organs into health." He thought, then, that the teacher should instruct the student in maxims of this kind, showing how empirical medicine mostly was, how treatment depended on prognosis, and how prognosis depended on shrewd observation of the ways of life and disease; and that, watching these ways of Nature, our attempts should be to modify them by such drugs, diet, regimen, and gymnastics as experience has suggested: that, in a word, we should endeavour to make medical students not merely trained men of science, but, that which is the end of their being—good clinical therapeutists.

Professor OSLER, F.R.S., said he thought the best contribution he could make to the discussion was to state what was the method of teaching practical therapeutics in the Johns Hopkins Medical School. They had the advantage of starting with a clean slate—without traditions in history. There was a three years' preliminary course, in which the men were taught the scientific branches. After two years in the medical school proper, the men entered the hospital at the beginning of their third year, and worked in the out-patient department and in the wards and in the clinical laboratories for two years.

They had a Professor of Pharmacology who was not specially interested in therapeutics, so that upon him, Professor Osler, devolved the duty of teaching the subject practically. The first important thing was to let the third- and fourth-year students see for themselves, week by week, the work of the clinic. Every Saturday there was presented to the class a sort of clinical summary of the work on two large blackboards in the class-room; the main features of the acute cases were presented, so that they had got composite pictures of such important diseases as pneumonia, typhoid fever, and malaria. It was very soon found to be impossible day by day in the wards to go into all the points essential in treatment, and it became necessary to supplement the teaching; and one of the assistants, Dr. Barton Jacobs, began a class on practical therapeutics. This plan was still further developed by Dr. Macrae as follows: In the third year a series of demonstrations were given, as on bleeding and the kind of cases suitable for it, also cupping and leeching. Hydrotherapy was taught to the third-year class. Once weekly, in what is termed a recitation class, drugs were considered systematically, the subjects being announced beforehand, chapters in the textbooks assigned, and special articles given out—a system which is an admirable substitute for the dry lecture. Often the students themselves were made to take part in the teaching by presenting special reports on drugs. There was a class in prescription writing. When in out-patient teaching a case was assigned to a student he was asked to follow the patient to his home, so as to have the opportunity of seeing the result of the treatment. The fourth-year students are divided into three groups, which take three and a half months each—medicine, midwifery and gynecology, and surgery. The routine work of the wards occupied all the morning. The time of the physician largely taken up with new cases, allows the student to pick up in an incidental way a good deal of interest relating to treatment, but he did not get it in a very systematic way. Therefore the teacher of clinical therapeutics took the group of fourth-year men, who were acting as clinical clerks in the ward, three times a week for special instruction. The action of drugs was fully considered, and, as each student had six or eight beds assigned to him, the effects of the drugs in his own cases were carefully watched. Special methods of treatment, such as hydrotherapy, were also considered, and also dietaries. And, lastly, the whole method of psychotherapy and Weir-Mitchell treatment was considered in suitable cases. This plan had succeeded admirably. He did not think it possible for all the points in practical therapeutics to be taught by the busy physician while on his rounds. He was constantly giving some instruction, but, unless it was done systematically, many things were sure to be overlooked. All the points upon which Sir Clifford Allbutt had dwelt were very important for the student to know before he went into practice, but he would rather concentrate the attention on teaching thoroughly two or three important diseases than cover a larger range incompletely. If a man were taught broad principles he would go out of hospital with wider ideas than if his time had been taken up with minutiae of prescriptions and therapeutics. The best basis

for a man's therapeutical knowledge was to recognize the natural history of the diseases he had to treat. That plan was infinitely to be preferred to the old method which was carried out in many schools, in which students had to listen to long courses of lectures in therapeutics.

Dr. HARRINGTON SAINSBURY desired to approach the subject from the point of view of the teaching of therapeutics in the medical curriculum. The way in which one came to the wards was all-important in making use of those wards subsequently. The importance of the subject was greater nowadays than ever because of the crowding of the pharmacy shelves with insufficiently tried remedies, and such crowding must increase since the introduction had become a commercial interest. The importance was greater also because present-day treatment was much more random than the treatment of the past. The extent to which the older physicians bled, purged, blistered and mercurialized was amazing, but there was virtue in that persistence because it was bound in the long run to declare itself for ill or good; whereas random treatment, the flitting from one remedy to another, could only lead to confusion, and the more so having regard to the large influx of remedies which had occurred. In respect of the position of therapeutics in the medical curriculum, it must in a sense be the last word to the medical student. All other teachings must converge upon treatment; but therapeutics must not wait to the last for the first words; they must begin with the preliminary studies. Medical studies were intimately associated throughout, and thus anatomy should always have an eye to the practical—for instance, the use of land-marks—and physiology also should always accentuate the facts which were therapeutically utilizable; whilst when one came to *materia medica* and pharmacology one found the subject so mixed up with therapeutics that there was great difficulty in dealing with it because of the stage of the curriculum in which these lectures came. From the beginning students could not avoid therapeutics, and should not if they could. The point, then, was that throughout the preliminary subjects treatment should be kept in view; and if this could be done, the practical side being made more prominent, he believed a great difficulty would be got over for a number of students, some of them born practitioners, who yet stumbled over the preliminary subjects because they did not see their practical bearing. Coming to hospital practice, it was obvious that therapeutics must engage the student's attention from the first post until the end.

Now with regard to *materia medica* and pharmacology, he would ask whether a few lectures on the history of medicine could not form part of the course. A strict science might be able to do without a history, because the laws could be always verified and demonstrated; but an art, a fortiori a great art, could not do without its tradition, since tradition meant the accumulated wisdom of the past, and medicine was much more an art than a science. He was convinced that we suffered seriously from the absence of teaching of the traditions of medicine. He could conceive of no better corrective to the commencing practitioner with a view to the avoidance of hasty theorizing and

10 Calvert: *Discussion on Teaching of Therapeutics*

ill-judged new (!) departures in practice than a knowledge of what had been done in the past. As men who aimed at being scientific, could we afford to do without the knowledge of the experience, and of the reflexion upon experience, of over 2,000 years of practice? Therefore he would say let retrospect always precede the new departure. Further, the student needed a brief statement or survey of the fundamental teachings which have present-day acceptance. Medical treatises were called "Principles and Practice," but he would ask where were the principles? The aphorisms of Hippocrates constituted one of the most celebrated books of antiquity. Those aphorisms embodied in concentrated form the wisdom of the past: they were the guiding principles of practitioners for many centuries. But was the present-day student ever taught to even look at them? They were apparently only for the curious, and by them they were regarded only as literature. He was sure they were at present on the wrong lines, and they would not get back to the right ones until they had got more into touch with the past. As to the best placing of the historical survey and of the summary of guiding rules in the curriculum of the medical student, he was not so clear as that we ought to have both. Whether they should form part of the *materia-medica* course, or should come in with the lectures on medicine might be a matter of debate.

The teaching of therapeutics in hospital wards came next, but he would leave the dealing with that subject to others. The essential thing was that at the bedside the practical element—demonstration—should always be foremost,

Dr. CALVERT said he thought that from the practical point of view they would improve the teaching of therapeutics in the wards if there were a little more independent prescribing, if the prescriber depended less on the hospital pharmacopœia. The hospital pharmacopœia was very valuable, and was absolutely necessary for the easier working of the dispensary. Still, at least in the wards, there might with advantage be a little more independent prescribing. The student should be taught how to prescribe more thoroughly than he was at the present time. The tendency at the present time was to send a man into practice chained to his hospital pharmacopœia, so that when at the bedside of a patient, having made his diagnosis, he determined from his pharmacological knowledge to use this and that drug, he could do so if the desired combination chanced to be in his hospital pharmacopœia; otherwise he tended to recline upon the tablets of the manufacturing chemists. These tablets, no doubt, were often very useful, but they did not necessarily give a man just what he wanted, nor the form in which he wanted it. And if he had not adequate knowledge, he was rightly afraid to prescribe on his own, for he was almost sure to come to grief over it, and he might do something dangerous. He had known men prescribe the *mistura hydrag. perchlor. cum pot. iod.*, which was found in every hospital pharmacopœia, for a syphilitic case, and then, to counteract the depressive action of the drugs, to have added *liq. strychninæ hyd. Mv*. Now this particular *mistura* by interaction contains a double iodide of mercury and potassium, and a double iodide is

an extremely well known precipitant of alkaloids; therefore all the strychnine is precipitated to the bottom of the bottle. That kind of thing was happening constantly, because men were not properly taught prescribing. In the dispensary at St. Bartholomew's, at the present time, they keep a special syrup of lemons made from the peel, for the following reason: Syrup of lemons of the British Pharmacopœia is a very popular flavouring agent; it is made from the lemon juice, and therefore contains much free citric acid. The house staff often forget the presence of the free acid and send in prescriptions of which the following may be taken as an example: Salicylate of sodium 10 gr., ammonium carbonate 5 gr., made nice with syrup of lemons. The acid effervesces with the ammonium carbonate and seizes the sodium of the salicylate, and the salicylic acid, being insoluble, is precipitated to the bottom of the bottle. Therefore, the dispensary keeps a syrup of lemons, made from the peel, which does not contain any free acid. There were many such simple things which were never taught to the students, but it was very desirable they should be. No one could prescribe independently with success unless he knew the common incompatibles. Some slight recognition of incompatibility ought to occur in examinations, as men could not be made to take much interest in anything which did not promise an examination reward; for this he did not blame them, as they were already overburdened with work.

Dr. R. HUTCHISON said he would like to deal with the subject from the point of view which Dr. Sainsbury omitted—namely, that of the hospital physician working in London—for although he had listened with admiration to the syllabus described by Professor Osler as obtaining at the Johns Hopkins Hospital, it was an admiration tinged with envy, and regret that it was an impossible system here. He believed it to be the case that the Johns Hopkins men were picked men, and he was sure the ordinary English student could not, with advantage, be put through a curriculum of that sort, even were there enough assistants to carry it out. So that one had to work on the old-fashioned lines. One question which had not been raised in the discussion, but a preliminary one of some importance, was as to the extent to which it was necessary to teach therapeutics to the student at all; for it might be argued that if one had made a correct diagnosis, the treatment ought to follow as a matter of course. Certainly if it were to be a fight between diagnosis and therapeutics, as to which should have most attention in the wards, he would lean to diagnosis. If one turned out a student who was not good at diagnosis, he was not likely to be successful in treatment. He was not saying that therapeutics should not be taught at all, but he believed, on deeper reflection, that it was not the important subject in the curriculum which many people imagined. Not only was it unnecessary, from one point of view, to teach therapeutics, but there was much in therapeutics that it was impossible to teach at all. He thought every practical physician would admit that the "power of healing" was a thing which could not be taught. Some doctors were good healers, others were not, and probably that was largely a psychical

question; it was a question of being able to bring one's personality to bear on the patient's personality, and constituted a great part of the healing art. This instinct for treatment could not be taught. If it came at all, it was by a large amount of practical experience in dealing with disease. In his own attempts to teach therapeutics in the wards he had found very great difficulty in getting students interested in the subject. The reason might be, as had already been suggested, that the subject was not made much of in examinations. He thought it was also true that the subject was not made enough of in the text-books of medicine which the student read. When that was so the student naturally regarded treatment as of comparatively little importance. But it was not only the question of examinations which made it difficult to interest the student in treatment; it was also due to the fact that unless a man was made actually responsible for treatment one could not expect him to be really interested in it. This lack of responsibility was at the bottom of much of the lack of interest in therapeutics amongst students. In Edinburgh the students attended dispensaries, where each was responsible for treating a certain number of patients in their own homes, though, of course, he had a chief in the background to whom to refer in cases of doubt or difficulty. Some such system was much wanted in all the medical schools of this country, which would give to the student the complete management of cases, and make him responsible, under supervision, for their treatment. The student, it had been said, should come to the wards with a good knowledge of pharmacology, but personally he would cheerfully dispense with that, except from the examination point of view. What he regarded as more important was pharmacy, a knowledge of the prescriber's armamentarium which he confessed the student did not now possess. In the wards one had largely to be content with teaching the technique of treatment. At the London Hospital there was a class which was taught by some of the senior sisters where students went to learn how to make beds, to give enemata, or matters which were often better understood by nurses than by practitioners. This class was found very useful. It was also important that students should study for themselves, as far as possible, the effects of drugs—not on the healthy body, as the pharmacologist did, but in disease. Observation of the effect of various drugs in cases of high blood-pressure was an example of what he meant. He thought, finally, that much of the want of success in teaching therapeutics was due to the lack of agreement among teachers themselves on the subject of treatment; it should not be possible, for instance, for a student to get confused because of the different methods of treating such a disease as pneumonia. It was surely time that a common plan of treating all the common diseases was agreed upon; its absence greatly perplexed students and tended to make them therapeutic sceptics.

Professor DIXON said that as the subject of pharmacology had received some criticism, he would like to say a word in its defence. It had been advocated there that *materia medica* should form an important part in the medical student's curriculum. He thought it was not generally appreciated that in

former times, say even fifty years ago, there were only two preliminary subjects taught to medical students—*anatomy* and *materia medica*—since these were the only two about which we had precise knowledge. Hence these were taught with extraordinary detail, and yet the medical student was not overworked. And now, when definite and precise knowledge of much more important subjects was available, we still allowed the old subjects to retain their prominence; we were too conservative; we adhered to *materia medica* though it contains matter of little or no practical use to the student of medicine. The chemist had long since taken over to himself *materia medica*. Dr. Calvert laid much stress upon the importance of prescribing, and especially of a knowledge of incompatibles. But surely lists of incompatibles were a remnant of the polypharmacy days; unfortunately, physicians still prescribed crude drugs like belladonna, which was a complex organic mixture, when they desired only to obtain the effect of atropine. Furthermore, even if the physician did prescribe together incompatibles it was the duty of the chemist to make the mixture compatible, and, indeed, some of their training had that object in view, though, of course, it was true that such prescriptions should not be written. The teaching of therapeutics in the wards was a demonstration of the application of pharmacology to the patient. Dr. Hutchison had just said that he did not greatly favour the student learning pharmacology, but later on he told us that when he got into the wards he should be taught the action of nitrites, digitalis, &c. But this was just the knowledge which was taught in the pharmacological laboratory, the only place where the action of the drug could be directly seen by the student himself. Each of his students, for example, had observed for himself the action of digitalis on the heart, of nitrites on blood-vessels and blood-pressure, of ergot on the uterus, of mercury on protozoa, and so on with the whole series of drugs. Book knowledge of drug action he regarded as of little value. Sir Clifford Allbutt, in his opening address, laid stress upon empiricism in the treatment of disease, and as an example quoted digitalis, which had come down to us as a folk-drug. That was true; but almost every plant that grew in the British Isles had at some time a reputation as a folk-drug. The majority of these have been thrown aside by the investigator, only a few of the poisonous ones being retained. Up to about forty years ago digitalis, and lead also, were generally regarded as circulatory depressants. It was the experimentalist who showed the mechanism of action of these drugs, results which were impossible to obtain at that time from clinical observation. Exact knowledge on the action of drugs could only be obtained except in rare cases from laboratory experiments, and he regretted that the application of laboratory facts had not entered more into therapeutics. As an example he might refer to the British Pharmacopœia as representative of the general ideas of medical practitioners, and there, to take a haphazard example, it was found that three or four preparations of opium were used for local application, though it had long ago been shown that neither opium nor its alkaloids had any local anæsthetic effect; the whole action was central. Many other examples might be given illustrating the want of enterprise of modern therapy.

14 Beddard: *Discussion on Teaching of Therapeutics*

Dr. BEDDARD said he agreed with Dr. Hutchison that it was unnecessary, if not impossible, to teach treatment. A man should come into the hospital wards having a good knowledge of the mode of action of drugs and the other measures used in therapeutics. In the wards treatment was a deduction from accurate diagnosis, from a knowledge of the effect that was required to be produced and from the means at our disposal for producing it; and it was in any given case either obvious or practically impossible. Treatment was necessarily guided by experience, which every man had to gain for himself and which would be taught to students only to a very limited extent. He considered that a knowledge of pharmacology was of great importance. There were many drugs whose mode of action was unknown; but there was also much treatment based upon erroneous ideas of pharmacology, and in text-books of therapeutics there were many so-called physiological actions which were unknown to pharmacologists and which were only explanations of actions supposed to be observed in treatment. Much medical treatment was empirical; but treatment was often called empirical which was really based upon unconscious diagnosis and a shrewd appreciation of the best line along which to treat the case. It was dangerous to advocate empiricism before students; such teaching could only foster the extraordinary credulity shown by many members of the medical profession. In face of the stream of new remedies with which doctors were being inundated by manufacturing chemists, it made one think that a critical spirit and a healthy scepticism were some of the greatest blessings which could be imparted to the medical student.

Sir DYCE DUCKWORTH said the various opinions which had been expressed in the discussion had interested him greatly. He believed that a great deal of therapeutic knowledge could be conveyed in the wards in the presence of sick people; that had been his opinion for years, and he had tried to carry out both therapeutical and clinical instruction. He had not been guilty of prescribing very constantly from the hospital pharmacopœia. He took infinite pains that his students and clinical clerks should learn to understand the prescriptions which were dictated to them; and it was the rule, when the medicines came up next day, to hand the bottles round to the class for the students to taste and smell the ingredients, the pills being broken up, and the liniments rubbed on the skin. They were also carefully instructed in making poultices and arrowroot, applying blisters and leeches, and understanding how long they should be kept on the skin. They were taught by the sisters how to make beds. Many of his colleagues did not undertake that kind of work, but he had the satisfaction of occasionally receiving gratifying references to it from old students in various parts of the world. Of course, those who had been longest in practice must know that a great part of the physician's work was empirical; but it was not base because it was empirical. He thought that Society was the place to lay down the fact that the profession nowadays, and especially the students in it, were mightily distracted by the enormous number of remedies which were foisted on to their attention from

week to week and month to month, chiefly from Germany, and that those things had a greater commercial than clinical value. But if the student and the practitioner believed in them they were likely to lose any knowledge which they might have acquired as to the exact nature and use of remedies. Our forefathers were not all fools; they were thoughtful men who observed and meditated; they did not live in a motorial age, like the present. There was a danger that the knowledge of the wise men of the past might be displaced by nonsense from the pharmaceutical laboratories at the present day, and he believed that that very strongly accounted for the wave of scepticism as to remedies which was evident in the profession to-day. If scepticism was present in the profession it was because the teachers were sceptical. The average student was now profoundly ignorant of his pharmacopœia, and he had generally found him very ignorant of his materia medica. Some had a greater liking for the study of drugs and medicines than others, but much of the sceptical state of mind and carelessness in prescribing was due to want of systematic teaching and the overlooking on the part of the physician in the wards to see that the prescriptions which he dictated were accurately written down, and because the results of the remedies were not carefully followed up. It was true that that took up more time, but hurried visits never did much good, and if the physician was to teach efficiently in a hospital he should not be in a hurry. Those who taught on the lines he had mentioned, and had faith in what they taught, would not fail to have successful therapeutic pupils.

Dr. HACKNEY (Hythe) said he had been a general practitioner forty-one years, and he felt very strongly that it would be a good thing if, when men left the hospital, they were to act as assistants to medical men before going into practice. There they would learn how to do many routine things: how to see and manage patients, and how to be business-like, including the efficient keeping of books, for records were very important. He was with his father for five years, and had to dispense, to keep books, and see patients. He recommended young men when they left the hospital to go into the country and work with some good general practitioner before settling down into practice on their own account.

The PRESIDENT (Professor Cushny) said the discussion had involved some criticism on the two sides into which therapeutics was divided—the laboratory part and the hospital part. He was himself a laboratory worker and found it very difficult to arouse any interest, either in students or in teachers, with regard to therapeutics. His experience of therapeutic teaching in the wards had largely been that the physician would make a careful diagnosis by the modern methods; and he would either say, "We will see how he is to-morrow," or "Give him a dose of calomel." If a dose of calomel were given, it was the last that was heard of the treatment; there was no apparent attempt to point out the action or the effects of the drugs. Apparently therapeutics was looked upon as something which was fixed, and there was very little more to work

16 Cushny: *Discussion on Teaching of Therapeutics*

upon. That was a sort of reflex from the school of Virchow, which taught that therapeutics was a hopeless sort of business and that the post-mortem was the thing to aim at. From that the present-day teachers were only slowly recovering. He felt very much that the new school of pharmacology could scarcely be blamed for the ignorance of the students, because thirty years ago the student had teaching of therapeutics in the wards and nothing else. Now he had got pharmacology in addition. If he knew still less therapeutics at the present time, it was not just to blame the laboratory teacher for his want of knowledge.

Therapeutical and Pharmacological Section.

November 2, 1909.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

The Physical Signs of Incipient Pulmonary Tuberculosis and its Treatment by Continuous Antiseptic Inhalations, with the Results in Thirty Cases.

By DAVID B. LEES, M.D.

THE object of this paper is to advocate an unusual method of treatment in the early, and especially in the earliest, stage of pulmonary tuberculosis. Incidentally it will be necessary to describe the earliest physical signs of a tuberculous infection of the lungs, which are inadequately and incorrectly described in the text-books.

The treatment most in favour at the present time for cases of pulmonary tuberculosis consists of absolute rest while pyrexia is present, abundance of fresh air, abundance of nutriment, and graduated exercise under medical supervision when pyrexia has ceased. It is certain that these measures have aided in the recovery of many. They place the patient under the most favourable circumstances for fighting his battle with the invading bacilli. But there is one obvious defect: they do not include any organized attack on the bacilli themselves. They place the patient under favourable circumstances, but they leave him to fight alone. Attempts have, indeed, been made to give actual assistance by the injection of tuberculin. But this treatment is by no means devoid of risk. If used at all, it can only be slowly, in carefully regulated minimal doses, and under constant observation of the opsonic index. It is very questionable whether such injections are ever justifiable

in the early stage of pulmonary tuberculosis, for the opsonic index is then constantly varying in consequence of the auto-inoculation produced by the local disease.

Attempts to combat the tuberculous infection by means of antiseptic substances, such as creasote or iodoform, given by the mouth, have attained very little success, and these drugs may produce gastrointestinal irritation. The idea of administering antiseptic drugs by the air-passages suggested itself to physicians thirty years ago. In 1877 Sir William Roberts claimed good results in phthisical cases from the use of antiseptic inhalation by means of a "respirator-inhaler" which covered the mouth only, and which he directed to be "worn for fifteen, thirty, or sixty minutes several times a day." Dr. Coghill also employed a similar apparatus, and laid stress on the necessity that the patient should practise inspiration by the mouth and expiration by the nose. "Each inhalation should last from fifteen to twenty minutes, and may be with advantage frequently repeated." The formula which he recommended in 1881 was this:—

R	Tinct. iodi ætherealis	5j
	Acidi carbolici	5j
	Creasoti vel thymoli	5j
	Sp. vini rect	ad 3j

and he also said that "where cough is urgent or breathing embarrassed, chloroform or sulphuric ether may be added at discretion." The quantity of the solution to be used was "ten to twenty drops twice a day at least," and he left the frequency of use largely to the decision of the patient himself. He adds: "A great many of my patients have of their own accord come to use the respirator almost continuously day and night, from their experience of its good effects."

Dr. Burney Yeo, in a lecture delivered in King's College Hospital in 1882, advocated a new and simple oro-nasal inhaler constructed of perforated zinc, and for use therewith he advised a mixture of "creasote, carbolic acid, eucalyptol, or turpentine, with equal parts of spirits of chloroform." He pointed out that such an inhaler is extremely cheap, that it is light and comfortable to wear, that patients find no difficulty in sleeping with it on, and that cough is rapidly relieved. He preferred a mixture of equal parts of creasote and spirit of chloroform, though he sometimes employed carbolic acid or eucalyptol, and occasionally iodine. He found turpentine useful in cases of hæmorrhage. He advocated "continuous or almost continuous inhalation." He stated that many cases under his care had improved remarkably by using this inhaler, and

he gave the particulars of one very striking case. But he does not claim that by the use of this method it is possible definitely to arrest the disease in its early stage.

Dr. Hassall, in his treatise on "The Inhalation Treatment of Diseases of the Organs of Respiration, including Consumption," published in 1885, endeavoured to prove that oro-nasal inhalers, and especially Dr. Yeo's, were practically useless; that a very small amount of the carbolic acid or other antiseptic used really found its way into the lungs; and that the only effective method was to place the patient in a chamber filled with antiseptic vapour. His objections, however, were founded on theory and on calculation, not on observation of clinical results. His inhalation-chamber is practically impossible in most instances, but his criticism seems to have been accepted by the profession, and to have produced a scepticism which resulted in an almost complete disuse of the method. Yet that excellent clinical observer, Dr. Wilson Fox, wrote: "The antiseptic effect has been doubted by Hassall, but there can be no question that inhalations practised in this manner, with creasote, thymol, eucalyptus, iodoform, iodine, or terebene, tend to diminish cough and expectoration, and that in some cases marked improvement in the patient's state occurs during their use, even in very advanced stages."

If a treatment by inhalation of antiseptics is to be really effective, three conditions are essential:—

- (1) It must be continuous and constantly in operation during the whole of the twenty-four hours, except at meal-times;

- (2) The inhaler employed must be light and capable of being worn both in the daytime and during sleep; it must not hinder respiration, and it should be cheap;

- (3) The solution employed should be as strong as possible, and it may with advantage be composed of a combination of several volatile antiseptic substances.

During the last four years I have treated all my cases of early pulmonary tuberculosis by this method, and the results have been so remarkable that I desire to bring them before the profession, in the hope that the method may be generally used. The inhaler employed has been the simple oro-nasal cage of perforated zinc advocated by Dr. Burney Yeo. It is worn over the nose and mouth, and is kept in place by elastic bands behind the ears; it contains a piece of sponge or felt on which the solution is dropped. The only precaution necessary is to take care that the edges of the inhaler, which rest on the skin, are

not wetted, lest the skin should be stained or made sore. The antiseptic solution which I have employed has been of this composition:—

R	Acidi carbolici	5ij	}	5j
	Creasoti	5ij		
	Tinct. iodi	5j		
	Spir. ætheris	5j		
	Spir. chloroformi	5ij		

Of this solution, six to eight drops are poured on the felt of the inhaler every hour during the daytime, and two or three times during the night if the patient is awake.

The odour of the solution is not unpleasant, and patients appear to derive great benefit from its use. Cough is rapidly relieved without any sedative or expectorant medicines, and sputum, if any, is more easily expectorated and is lessened in quantity. The use of this solution has no irritating tendency, nor does it cause hæmoptysis. If hæmorrhage should occur, it might be well to remember Dr. Yeo's suggestion and to add turpentine to the solution. The absolutely continuous use of the inhaler (except at meal-times) must be rigidly required, and it is very desirable to keep the patient at rest in bed for a week at least, the windows of his bedroom being widely open. During the second week he may be allowed to rise for an hour or two daily, but the continuous use of the inhaler is essential. When the temperature is normal, after the first ten days or so, he may be allowed to omit the inhaler for an hour every morning and to take a walk in the open air. Gradually the periods of exercise may be increased and the number of hours during which the inhaler is used may be very gradually diminished.

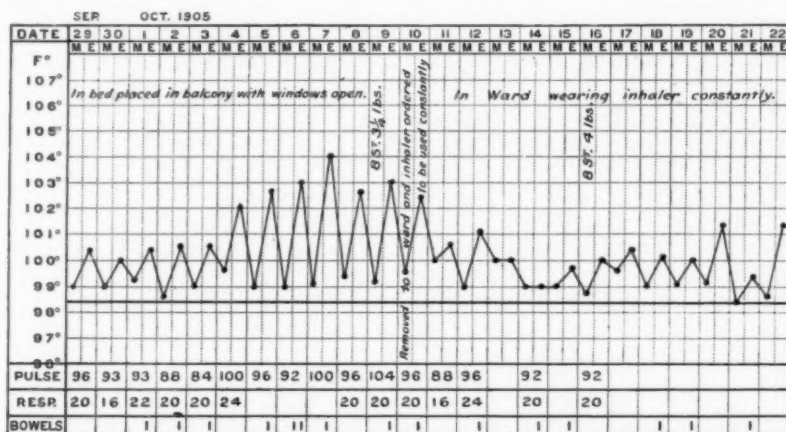
Two other points claim attention from the outset. The first is the condition of the teeth, gums, and throat. After every meal the mouth should be rinsed with sanitas fluid and water in equal parts. As soon as possible, decayed stumps of teeth should be extracted and carious teeth filled or removed. If the tonsils or fauces are unhealthy, they may be sprayed with a solution of perchloride of mercury of the strength of 1 in 2,000. If there is any nasal obstruction it is advisable to spray the nostrils thoroughly two or three times daily with this solution:—

R	Cocain	gr. iv
	Ol. eucalypti	ʒss
	Parolein	ʒj

by means of a parolein spray-apparatus. If the patient is a smoker, it is necessary that he should give up the use of tobacco at once and permanently.

The second point which claims attention is the question of nutriment. It is essential to administer a sufficiency of easily-digested and nutritious food. I insist on every patient taking four times daily, in addition to his ordinary meals, a half-pint of (scalded) milk to which from a dessertspoonful to a tablespoonful of "malted milk" has been added.

Before relating the thirty cases of incipient or early pulmonary tuberculosis which I have treated by continuous antiseptic inhalation, I exhibit a temperature-chart taken from a case of advanced phthisis



Temperature chart of W. C., case of advanced phthisis.

in which the employment of this method caused an immediate reduction of the hectic temperature, with arrest of sweating and slight gain in weight.

W. C., aged 32, was admitted into St. Mary's Hospital during my autumn holiday in 1905. When I resumed charge of the wards in October I found his bed placed on a sheltered balcony, with open windows to allow continuous fresh air as fully as possible. Removal from this to a hospital ward, with the use of continuous antiseptic inhalation, produced the striking effect shown by the chart. The course of the disease became much less acute, and finally so chronic that it was necessary to transfer him to an infirmary.

The following cases have been treated by this method:—

(1) I. B., aged 19, was admitted into St. Mary's Hospital under my care on March 8, 1907, with signs of early tuberculous affection of both upper and lower apices of both lungs and evidence of fluid effusion into the left pleura. There was persistent pyrexia, the temperature being between 101° and 102° F.; twice in the first week it fell to 99° F., and once rose to 103° F. The antiseptic measures I have mentioned were begun at once, and she wore the inhaler day and night. There was no sputum. The pulse-rate varied from 84 to 100, the respiration-rate from 28 to 36. On March 17, 33 oz. of clear fluid were aspirated from the left pleural cavity; this fluid was found to be sterile. On March 18 her weight was found to be 8 st. $10\frac{1}{2}$ lb. The antiseptic measures were continued. During the second week her temperature varied from 99° to 101° F.; it first fell to normal on March 23. It was a little irregular from March 25 to March 27, and again from April 2 to April 7, but after this it remained persistently normal. On March 27 her weight was only 8 st. $5\frac{1}{2}$ lb., showing a loss of 5 lb. in nine days. From March 27 to April 6 she had diarrhoea, so that I feared that she might have tuberculosis of the intestine, and her weight fell to 7 st. 13 lb.—a total loss of $11\frac{1}{2}$ lb. But the inhaler had been worn persistently day and night from the first, and now came the turn of the tide. After April 7 the temperature remained normal and she began to improve rapidly. On April 10 her weight was 7 st. $13\frac{1}{2}$ lb.; on April 18, 8 st. $6\frac{1}{2}$ lb.; on April 25, 8 st. $11\frac{1}{2}$ lb.; on May 1, 9 st. $2\frac{1}{2}$ lb.; on May 6, when she was discharged, 9 st. $5\frac{1}{2}$ lb.—a gain of 20 lb. in twenty-six days. The difference in her appearance was extraordinary. One would hardly have known her to be the same patient.

(2) Mr. S., a medical student, was under my care in St. Mary's Hospital for several weeks in the summer of 1906. He had pyrexia, cough, and marked signs (with crepitant sounds) at all four apices. On two separate occasions the sputum was found to contain large numbers of tubercle bacilli. His opsonic index was found to vary from 0.9 to 1.2. He was kept at rest in bed, an ice-bag was applied over the affected areas of lung, and the antiseptic measures described above were employed at once, the inhaler being worn night and day from the first. His temperature soon fell to normal, his physical signs improved, and before he left the hospital he had gained 2 lb. in weight. Though the first and second examinations of his sputum showed large numbers of bacilli, on the third examination they were rather difficult to find. He now spent several weeks at Llandrindod Wells, wearing his inhaler most conscientiously, living as far as possible in the open air, and taking plenty of milk. When he returned to London in October he had gained 20 lb. in weight. His sputum was very scanty, but it still contained a very small number of bacilli. His improvement was so marked that I decided that it was unnecessary for him to go to Davos for the winter, as he was willing to do, and that he might gradually resume work. This he did cautiously, and with excellent success. His weight continued to increase, and on March 16, 1907, it was 11 st. $1\frac{1}{2}$ lb.—

a total gain since his illness of 30 lb. At this date he was still using the inhaler about an hour daily: he had recently passed safely through an attack of influenza. This gentleman still continues in excellent health, looks extremely well, and has done hard work and gained distinction in his hospital classes.

(3) Mr. E., aged 34, was seen by me in consultation with Mr. Jessett, F.R.C.S., and Dr. English on June 17, 1907. Five weeks previously Mr. Jessett had removed a chain of caseous tuberculous glands from the right cervical region. The wound healed well, but two days after the operation the temperature rose gradually and became of hectic type. The pyrexial period lasted seventeen days. For six days the temperature was nearly normal. This was followed by another pyrexial period of ten days; it was now again normal. The pyrexia was accompanied by increasing dyspnoea, he was expectorating thick, mucoid sputum, and some night-sweats were present. No tubercle bacilli could be found in the sputum, but the physical signs were quite typical of a tuberculous infection of both lungs. The patient was at this time in a nursing home in South London, but he determined to go back to his lodgings near Westbourne Park Station. I saw him again, in consultation with Dr. Thoresby Jones, on June 29. He occupied two rooms on the first floor in a rather narrow street on a motor-omnibus route—by no means an ideal place for the treatment of such a case. His bed was in the back room; the folding-doors were thrown back, and both windows of the front room were constantly widely opened. He was fortunate in obtaining the help of a most excellent nurse. It was found that the physical signs had considerably increased during the twelve days, and there was now a pleural effusion on the right side. This fluid was drawn off and proved to be sterile. After the aspiration the side was strapped and an ice-bag applied. A Yeo's inhaler was worn constantly day and night, in accordance with the method already described, and he was fed almost exclusively on milk and malted milk. The sputum increased rapidly in amount and became mucopurulent, the temperature continued at 102° F., and there was discomfort about the larynx. An ice-bag was applied over this region and two others over the lung. The very copious and purulent sputum was examined bacteriologically, and was found to be full of streptococci. This fact, indicating a streptococcal infection of a tuberculous lung, seemed to me to render the prognosis almost hopeless. On July 11 a very violent paroxysm of coughing occurred, lasting five or six hours, when he brought up much purulent material and some blood. The temperature rose to 103° F. and the pulse to 130 for a short time; but this soon subsided, and he seemed better. He was assiduously nursed and carefully fed, and his appetite began to improve. The inhaler was worn persistently. From June 26 to July 4 the temperature was from 100° to 102·3° F.; from July 4 to July 10, 99·5° to 101° F.; from July 10 to July 14, 99° to 100° F.; and on July 14 it became normal permanently. On July 23 he was still improving, and was obviously gaining flesh; sputum much less, but still copious and mucopurulent; one or two slighter paroxysms of coughing. There was now resonance in the right axilla (where the fluid had been), but an area of dullness

existed at the right anterior base and the right posterior base, with some bronchial breathing and moist sounds—clearly a basic cavity. On August 16 he had sufficiently recovered to come to my house, and I found that there were still signs, though less marked, of a cavity at the right base. He looked decidedly stouter. His weight was found to be 11 st. 10½ lb., his weight in health having been 12 st. 6 lb. He was sent into the country, and persisted in the use of his inhaler. After this his recovery was uninterrupted, and he resumed his work. I examined him again on April 28, 1909, after an interval of a year and eight months. All the old dull areas could be detected, but there was no bronchial breathing, no prolonged expiration, and no crepitant sound—in short, no sign of activity. His weight had increased to 15 st. 6 lb.—an increase of no less than 52 lb.! He looked so well that I hardly recognized him at first sight. He had done full work for more than eighteen months, from 9 a.m. to 7 p.m. each day, in an ill-ventilated shop, and had enjoyed a summer holiday in France.

These three cases give sufficient proof of the curative influence of continuous antiseptic inhalation in a tuberculous, and even in a streptococcal, invasion of the lungs. They justify the hope that if this treatment can be adopted in the earliest stage of a pulmonary tuberculosis, we may be able definitely and completely to arrest the disease. If we are to have any prospect of abolishing pulmonary phthisis—one of the greatest scourges of the human race—we must attack it long before any bacilli can be found in the expectoration. In the great majority of cases of pulmonary tuberculosis, definite and characteristic physical signs are present for weeks or months before the patient begins to expectorate. To wait for tubercle bacilli in the sputum before diagnosing pulmonary tuberculosis is like delaying the diagnosis of cancer until the glands are involved. It is therefore essential to acquire accurate ideas as to the exact localization in the lung of an incipient pulmonary tuberculosis and as to the physical signs which are caused by it. For a statement of the current teaching on this subject I turn to the latest authoritative monograph, the article on "Tuberculosis of the Lung" in "Green's Encyclopædia and Dictionary of Medicine and Surgery," vol. vi, published two years ago. In this article, written by a physician of wide experience, there is a section on page 35 under the title "Physical Signs of Incipient Tuberculosis," which states that "The early determination of pulmonary tuberculosis by physical signs affords scope for great refinement of methods. In proportion to the earliness of the process, the physical signs are slighter they are, consequently, apt to be missed. Reliable skill can only be obtained after prolonged and careful practice. *Auscultation* affords the

more delicate tests. The auscultatory phenomena include slight modifications of the normal respiratory murmur, *e.g.*, enfeeblement, harshness, interrupted or cogwheel or jerky respiration, prolonged expiration, blowing expiration (broncho-vesicular). When the respiration is actually bronchial, the process is something more than incipient merely. . . . "*Percussion* yields results of importance even at early stages. All portions of the lung must be examined. Corresponding portions must be compared with care; more especially the two apices must be tested comparatively in respect of percussion sound and extent of resonant area."

This statement as to the earliest physical signs in an incipient pulmonary tuberculosis, and similar statements in the text-books, seem to me both erroneous and very inadequate—erroneous in assigning greater importance to auscultation than to percussion, and quite inadequate in the information given as to the regions of the thoracic wall where we may expect to find the earliest changes. Yet it is now nearly twenty years since Dr. Kingston Fowler pointed out that tuberculosis of the lungs begins, in an overwhelming majority of cases, at certain definite localities in the lung, and that the disease progresses by well-defined routes. This statement was founded on Dr. Fowler's experiences of autopsies at the Brompton Hospital. It has found its way into some of the text-books, and is recognized as conveying true information of pathological fact; but it has produced little or no modification of the descriptions of the clinical picture of the disease. Dr. Fowler showed that the earliest lesion occurs not at the very summit of the lung, but at a spot about 1 in. to $1\frac{1}{2}$ in. below the summit, in the lung as seen in the post-mortem room. This will correspond to a greater distance, probably quite 2 in., in the expanded air-containing lung. From this site the process may extend either backwards towards the supra-scapular fossa, or downwards and outwards into the second intercostal space. Clinically this can quite easily be recognized by careful percussion. The earliest physical signs (dullness and lessened air-entry) may usually be first detected in the innermost part of the first intercostal space on one or both sides, extending from one to three fingerbreadths from the sternum, and possibly also in the second space at about one fingerbreadth from the sternum. Posteriorly they may be detected quite close to the uppermost dorsal vertebræ on either side, a region which normally is quite resonant. Dr. Fowler also pointed out that less commonly the site of the earliest lesion was situated more externally, and this also is recognized clinically by dullness in the

outermost part of the first, and sometimes of the second, intercostal spaces; posteriorly it can be detected in the outer part of the supra-scapular fossa, occasionally also in the upper axilla. This area of dullness often exists along with that first described, while the mid-clavicular region remains much more resonant.

In the lower lobe also a similar tuberculous focus is apt to be developed at a very early stage, with, or very soon after, the focus in the upper lobe. Dr. Fowler stated that in the lower lobe also the situation of this early lesion is about 1 in. to $1\frac{1}{2}$ in. below the summit of the lower lobe, and that it is apt to spread downwards and outwards along the lower edge of the interlobar fissure. He described the position at the back of the chest where this lesion was to be looked for. Clinically it is quite easy to verify these facts if careful percussion is practised over the inner extremity of the spine of the scapula and the adjoining region.

These areas at the four apices are the most frequent and characteristic, but occasionally small areas of dullness may be found in front in the right middle lobe, and in the antero-lateral region of the anterior base of the left lung; posteriorly, at the posterior scapular edge, at the scapular angle, or near the posterior base of the lung. The existence of dullness and lessened air-entry at these various localities is quite easy to detect. All that is required for their discovery is care, patience, a correct method of percussion, and a little practice. Anyone who is willing to take a little trouble can satisfy himself of the accuracy of what I have stated. Over these dull areas auscultation often reveals nothing but a defective air-entry, though very slight crepitant sounds may be present: such sounds may or may not be removed by a cough. At a slightly later stage the inspiration may be a little harsh or interrupted; the expiration slightly prolonged. When bronchial breathing, bronchophony, and distinctly audible whisper are present, there is either consolidation around a bronchial tube or a definite cavity.

I may point out the similarity of the earliest physical signs in pulmonary tuberculosis to the earliest physical signs in pneumonia. On the first day of a pneumonia, before any sharp crepitation or any bronchial breathing is developed, it is often possible to detect by careful percussion a small area of diminished resonance or actual dullness, over which the only auscultatory sign is deficiency of air-entry. In both tuberculosis and pneumonia the similar phenomena are due to a similar cause, a local microbic invasion and a local arrest of pulmonary function.

The typical localization of these areas of dullness and lessened air-entry at the four apices is, in itself, almost proof of a tuberculous infection

of the lung, but in young subjects they may be simulated (though not often exactly) by simple collapse, and occasionally by broncho-pneumonia or by enlarged bronchial glands. They are not necessarily proof of an *active* tuberculous lesion; they may be the abiding result of a former lesion which has recovered or become quiescent. Local crepitant sounds over these dull areas, pyrexia, hacking cough, hæmoptysis, or local tenderness, would indicate an active lesion. These physical signs are the earliest obtainable evidence of the existence of pulmonary tuberculosis. They are long antecedent to the appearance of any sputum, and therefore to any possibility of microscopical examination. They are also quite distinct long before any evidence can be obtained by the use of Röntgen rays: the statements which have been made to the contrary are founded on inaccurate percussion. But a variability of the opsonic index may exist along with the earliest pulmonary signs: of this I have twice had proof. A worker in the bacteriological laboratory was found to have a varying opsonic index to tubercle, though he felt quite well. He came to me and requested me to examine his lungs. I found minimal areas in the characteristic positions, measuring from a half to one fingerbreadth—i.e., 1 to 2 cm. He did not practise inhalations, but went to a sanatorium, where two months later he had a pyrexial attack lasting a week, and a few tubercle bacilli were found in his (very scanty) sputum. He remained in the sanatorium four months, and gained 8 lb. in weight. I examined him again on his return, and found all the dull areas distinctly larger but quiescent, and he seemed to be in good health. The second case is the converse of the first. A patient who had had a brief attack of pleurisy on the left side, and who quickly recovered, was found to have minimal characteristic areas in the left lung only. There was also slight enlargement of cervical glands. An investigation of the opsonic index to tubercle was suggested and carried out; it was found to be varying. The variability fortunately lasted only a few days.

The dull areas of pulmonary tuberculosis differ much in size. They may be detected when they measure only half a fingerbreadth, or 1 cm., but they often measure about two fingerbreadths. In a severe case they may reach three fingerbreadths, and as much as four in the latest stages of the illness. The auscultatory signs will be determined by the amount of implication and destruction of the pulmonary structure. The possibility of accurate measurement by percussion makes it easy to construct a rough diagram showing the localization of these areas, and the number of fingerbreadths in each can be recorded by figures. Thus, the condition at one visit can be compared with that found at a

subsequent visit. The areas may be found to be smaller when the case is improving, and larger if his disease has progressed. I find these records quite trustworthy, and of the greatest assistance in helping to determine whether improvement or deterioration has occurred.

I now add a brief description of twenty-seven other cases of incipient or early pulmonary tuberculosis treated by the method of continuous antiseptic inhalation :—

(4) Mr. M., aged 33, seen with Dr. Mowll, of Surbiton, July 3, 1905. After a sharp attack of influenza he was found to have signs of tubercle at all four apices. Loss of weight 7 lb. Recovered under constant use of inhaler, and gained weight, 14 lb. Went back to work, and in December, 1906, was examined for life insurance and accepted as a "first-class life." But in January, 1907, he had a fresh attack, chiefly in the left lung. He was again kept at home for a fortnight, and used his inhaler persistently; again regained the weight lost, and resumed his occupation, which involves both mental and physical strain. He has remained quite well and is fully equal to his duties.

(5) D. B., age 1 nearly 13, the son of a medical man, had been sent home from boarding-school because ill. Tall, but $2\frac{1}{2}$ lb. under average weight for age. Short cough. Signs (November, 1906) at all four apices. Under continuous antiseptic inhalation his cough vanished in a few days; he gained 6 lb. in weight in five weeks, and looked better than for two years. Advised still to use the inhaler three hours daily. He continued to improve; a year later he had gained 14 lb. more, and a year later still an additional 9 lb. In November, 1909, Dr. B. writes: "My son Douglas keeps well, loses no time at school, enjoys the games, and writes home for large cakes to be sent."

(6) Miss V., a hospital sister. Signs in left lung only at first, later at all four apices and one lower area. Loss of weight, 7 lb. No sputum. Weight gained in first three weeks of treatment, $3\frac{1}{2}$ lb.; in the next month, 5 lb.; total gain, $8\frac{1}{2}$ lb. Recovered completely, and for eighteen months has fulfilled her arduous duties with conspicuous ability and success.

(7) Miss M., M.B.Lond. Signs at all four apices. Weight lost, $4\frac{3}{4}$ lb. No sputum. Weight gained in first three weeks of treatment, $2\frac{3}{4}$ lb.; during the next month, $12\frac{1}{2}$ lb.; subsequent gain, 15 lb.; total gain, 30 lb. (Her weight rose from 114 lb. to 144 lb.) She has been quite well for two years, and is now doing school-inspection work six hours daily for five days a week; is giving ambulance lectures, and has private practice.

(8) Miss M., aged 28. Signs in left lung only at first, afterwards at all four apices and four lower areas. No sputum. Had lost weight. During first three weeks of treatment lost $\frac{3}{4}$ lb., and afterwards regained it; during the next month gained another pound. She then felt much better, and her cough had disappeared. I have heard from her recently that she is still well.

(9) Admiral X., aged 50, seen in consultation with Dr. G. A. Simmons, of Westminster, April 11, 1907. Eight months previously a little pleurisy on left side; four months ago another attack on right side, with a little hæmoptysis. Yesterday, again pain in side and slight hæmoptysis. Signs of tubercle in right lung only. Under continuous antiseptic inhalation he rapidly recovered, and in June, 1908, was "in excellent health" and doing important public work.

(10) Mrs. R., aged 38. Signs at all four apices, and a small cavity at left lower apex. Sputum mucoid. No tubercle bacilli found. Weight gained in first three weeks of treatment 4 lb., and during the next month 8 lb.; total weight gained, 12 lb. Cough and sputum much less. Looks much better. I have not heard from her during the last two years.

(11) K. S., aged 17, housemaid. Signs at all four apices; subsequently developed a small cavity at right apex. No sputum. Her weight was not taken until after six weeks' treatment; it was then 97 lb. She gained 6 lb. Recovered and remained well for six months, then caught fresh cold from a wetting, and developed a few fresh signs. But two months later she was "much better."

(12) Mr. C., aged 25, brought by Dr. G. A. Simmons, of Westminster. Four years previously, after an attack of influenza, was ill for three weeks with pyrexia of hectic type. Recovered slowly, and after this had cough each winter, and some sputum with occasional streaks of blood. Loss of weight, 7 lb. Frequent short cough. Tubercle bacilli not found in sputum. Signs at all four apices and two lower areas. Under continuous antiseptic inhalation his cough ceased in a week, and his first remark on his second visit was: "I can't think where my cough has gone to!" Gain of weight in first three weeks of treatment, $2\frac{1}{2}$ lb.; during the next month, $4\frac{1}{2}$ lb.; subsequent gain, $2\frac{1}{2}$ lb.; total, $9\frac{1}{2}$ lb. He spent some months of the winter of 1907-8 at a high altitude station in the Rhone Valley, under the observation of Dr. Tidey, of Montreux, using his inhaler six hours daily and at night, and returned to England in excellent health. I have recently heard from him that he has gained 7 lb. in weight since I last saw him.

(13) Mr. P., aged 36, sent by Dr. Wilson, of Boston. Signs at all four apices and four lower areas. Sputum scanty. Tubercle bacilli not found. Has lost 2 lb. in weight during the last three weeks. Under inhalation treatment he gained 5 lb. in four weeks. He continued to improve, and six months later was quite well, except for some chronic nasal obstruction. He has remained well.

(14) H. C., aged 19, brought by Dr. Hickley, of South Lambeth Road. Slight hæmoptysis two years ago, with signs at right apex. Two more attacks of slight hæmoptysis recently. Signs at all four apices and five lower areas. Under inhalation treatment he gained 1 lb. in the first three weeks. He used his inhaler for some months, and a year after his first visit to me Dr. Hickley reported that he had been "quite well for some time." I have recently heard that he continues well.

(15) K. P., aged 15, sent by Dr. Brooks, of Felixstowe, under whose care he had been for a month on account of pleurisy at the right base. His illness began two months ago. He had improved in general condition, having gained 7 lb., but the physical signs were stationary. A sister of his father's had died of phthisis after an illness of only ten weeks. His weight when he came to me on September 18, 1908, was 9 st. 7 lb.; he showed signs of tubercle at all four apices and five lower areas, including two large ones at the right base, but apparently no pleural effusion. The treatment by continuous antiseptic inhalation was adopted at once, under the supervision of Dr. Baggins and Dr. Adams, of Hartley Wintney, Winchester, Hampshire, and a very thorough open-air treatment was at the same time carried out by them, the boy living in an open shelter in his father's garden. Sputum very scanty. Twelve days later Dr. Baggins wrote that the cough was almost gone, and that he had gained 7 lb. in weight. In another month he gained 10 lb. more, and subsequently 3 lb.; total gain, 20 lb. In December, 1908, he looked fat and well, and had not coughed for a long time. His weight was then 10 st. 13 lb. In November, 1909, Dr. Baggins writes that he had recently examined the boy's chest very carefully, and could find no evidence of any disease of the lungs; he considered him cured.

(16) Mr. H., aged 20, a patient of Dr. Nettell, of Shirebrook, Mansfield. He had no cough, but had suffered from repeated attacks of tonsillitis. Four years previously he had suffered from pleurisy; he had lost 3 lb. in weight; he had signs at all four apices and four lower areas, the largest being at the right lateral base—doubtless the remains of the former pleurisy. No sputum. Under the antiseptic treatment by inhalation and sprays to throat and nose he rapidly improved, gaining $3\frac{1}{2}$ lb. in weight. He returned to his work as a mining engineer, and he continued well. It is doubtful whether some of his dull areas were not of old standing, the legacy of his attack four years previously, but they appeared to diminish slightly while he was under treatment.

(17) A. W., aged 26, marine engineer; sent by Dr. Beaumont, of East Ham. He had had cough for four months, and had been taken ill on shipboard. He was admitted into hospital at Sydney, Australia, and remained there ten weeks. He had been back in England for one week; he showed signs at all four apices and five lower areas, with a cavity at the right apex; he looked very ill, and had lost more than 3 lb. in weight. Under treatment by antiseptic inhalation he rapidly improved, and when I next saw him, four months later, he had gained $15\frac{1}{2}$ lb. in weight. Three months later still there had been a further gain of 3 lb.; total gain, $18\frac{1}{2}$ lb. He looked quite a different man, and had very slight cough in the morning only. There were still signs of a cavity at the right apex. I have heard no more of him.

(18) C. G., aged 40, had suffered from occasional slight attacks of hæmoptysis since 28 years of age, with slight cough. When he came to me on February 14, 1909, he had had a "cold" for five weeks, followed by an attack of hæmoptysis more severe than ever before. He showed signs at all four apices and two lower

areas, with possibly a small cavity at his right apex. After a week in bed, with continuous antiseptic inhalation, he returned to his work. He was able to wear the inhaler in his office for seven hours daily, also at night. During the first three weeks of treatment he gained $3\frac{1}{2}$ lb.; during the next month, 3 lb., and subsequently 2 lb.; total gain, $8\frac{1}{2}$ lb. His friends, he stated, "never saw him look so well." After giving up the inhalations he had a small extension of physical signs in the right second space, and lost $1\frac{1}{2}$ lb., but a return to the treatment soon arrested this. He is now in excellent health and doing full work.

(19) Miss T., aged 18, sent by Dr. Wilson, of Boston. Cough for three months, but no sputum. Signs at all four apices and two lower areas. Under treatment by continuous antiseptic inhalation the cough soon disappeared, and she gained $2\frac{1}{2}$ lb. in weight. I heard last month that she was very well "and growing quite fat."

(20) Miss H., aged 22, sent by Dr. Leeson, of Twickenham. Signs at first only in left lung posteriorly, but afterwards at all four apices and two lower areas. She had lost $2\frac{1}{2}$ lb. in the last three weeks. Under treatment by continuous antiseptic inhalation there was steady improvement; the cough rapidly diminished, and she told me that "life was now worth living." She gained 2 lb. in the first three weeks; $1\frac{1}{2}$ lb. during the next month, $2\frac{1}{2}$ lb. subsequently; total gain, 6 lb. Is still using the inhaler, and is "wonderfully better" and very active.

(21) Mr. B., aged 36, sent by Dr. Grove, of St. Ives, Hunts. His illness began eight months previously, with catarrh and hæmoptysis. Dr. Grove found signs of tubercle at the right apex, but no bacilli in the sputum. Cough worse for four months. Had lost 4 lb. Dr. Grove found tubercle bacilli for the first time the day before the patient came to me. There had been phthisis in his mother's family, and his father died of rheumatic fever. The patient had had pains in his knees, and was found to have a moderately dilated heart, a systolic apex murmur, and a short presystolic. He had signs of tubercle in his lungs at all four apices and two lower areas. After two weeks' rest in bed and continuous use of the inhaler he was able to return to his office, wearing the inhaler while at his work, and altogether about twenty hours out of the twenty-four. During the first three weeks he lost $\frac{1}{2}$ lb.; but in the next month gained $4\frac{1}{2}$ lb., and subsequently $14\frac{1}{2}$ lb., a total gain of $18\frac{1}{2}$ lb. He is now doing full work without fatigue, and gradually lessening his use of the inhaler. He feels very well.

(22) Mr. P., aged 38, sent to me by Case 3. Cough for four months. Felt ill and unequal to his work for one month. Signs at all four apices and four lower areas. No sputum. After a week's rest in bed with continuous antiseptic inhalation he returned to his work, and was able to wear his inhaler for several hours daily, and at night. The cough disappeared within a week. Gain of weight in first three weeks, 4 lb.; during the next month, 5 lb.; total gain, 9 lb. During July he had to work twelve hours a day, and he lost ground a little,

but he persevered with the inhalations, and by September had quite recovered. He was doing his full work without fatigue, and had had no cough for three months.

(23) Mr. H., aged 32, also sent by Case 3. Illness began four months previously, and he lost weight rapidly. He also suffered from hydrocele, which was tapped several times. Temporary improvement occurred, but a fortnight ago he went to Germany for a week and experienced "shocking weather." Came back feeling ill. Showed signs at all four apices and two lower areas, with a cavity at his right apex. Evening temperature $99^{\circ}8'$ F.; sputum scanty, but containing numerous tubercle bacilli. A week after his first visit to me he had hæmoptysis twice, and his temperature rose to 103° F. I saw him again six days later at his own home, in consultation with Dr. Rudd, of Acton. The temperature had fallen to 101° F. I found a quite new dull area above the left nipple, with sharp crepitant sounds, and the former physical signs were more marked. Evidently the disease was progressing rapidly; but under persistent antiseptic inhalation he improved more than we had dared to hope, the temperature fell to normal, and remained normal. He kept his bed for five weeks. There was no more hæmoptysis, and the cough became much less. He gradually increased in weight, and had gained 5 lb. in the eight weeks in spite of his severe attack. The physical signs were more advanced, and in addition to the cavity at his right apex there were signs of cavitation in three other situations. He was gaining strength, and was walking about two miles daily; cough not troublesome, still some sputum. He was advised to go to Bournemouth, and to wear his inhaler six hours daily, and at night. I have not heard from him again, but I have heard indirectly that he is purposing to emigrate to South Africa, and that he has grown so much stouter that he has been compelled to have his clothes enlarged.

(24) Miss M. R., aged 13, the daughter of a medical man (deceased), had suffered from a "tuberculous finger" at six months of age; it was opened surgically, and healed slowly. For about a year she had had cough and some pain in her side. Sputum scanty, and did not contain bacilli. Signs at all four apices and four lower areas. Under the use of continuous antiseptic inhalation her cough ceased in a week, and in six weeks she gained 14 lb. in weight. I saw her again five months later, and found a further increase of 11 lb.; total gain, 25 lb. She looked and felt extremely well.

(25) Mr. A., aged 37, brought by Dr. Higgins, of Hornsey Road. Cough five weeks; loss of weight, 7 lb. Signs at all four apices and four lower areas, with a cavity at the right apex. No sputum. Under treatment by continuous antiseptic inhalation he improved steadily. Gain of weight in the first three weeks, $5\frac{1}{2}$ lb.; during the next month, 5 lb.; subsequently, 7 lb.; total, $17\frac{1}{2}$ lb. His employers having generously allowed him a complete holiday, he was able to use the inhaler during nearly the whole of the twenty-four hours for the period of five months. When last examined, on August 4, he was perfectly well, the cavity having become obliterated. Since then he has gained $2\frac{3}{4}$ lb., and for three months has done his full work without fatigue.

(26) Rev. W. W., aged 42, a patient of Dr. Wilkin, of Newmarket. Ill eight weeks; loss of weight, 4 lb. Hæmoptysis ten days ago. Two brothers had died from phthisis, aged 38 and 48. Signs at all four apices and three lower areas. Under treatment by continuous antiseptic inhalation, he gained 9 lb. in the first three weeks, and 11 lb. during the following month. He improved rapidly, and when examined on October 13 there were no active signs in his lungs, and he had gained 6 lb. more; total gain, 26 lb. Was doing full clerical duty without fatigue.

(27) Miss S., aged 34, sent by Dr. Havell, of Felixstowe. Aching pain in her right chest, with sense of constriction and some dyspnœa, for nine months previously; worse during the last six weeks. Very slight hæmoptysis. Signs at all four apices and two lower areas, with a cavity at the right apex. No sputum. Under antiseptic inhalation, continuous so far as her duties would allow, she has slowly and steadily improved. She has gained $3\frac{1}{2}$ lb. in weight, her cavity has slowly decreased, there has been no cough for some time, and only occasionally any pain. She is doing her full share of work without fatigue.

(28) C. H., aged 9, the ill-developed and half-starved child of very poor parents, living over a stable, was found on April 30, 1909, to have signs of tubercle at both apices of the right lung and at the upper apex of the left. His weight was only 3 st. $6\frac{1}{2}$ lb., or 12 lb. below the average weight for his age. He had enlarged cervical glands and many carious teeth. My colleague, Dr. Willcox, kindly admitted him into St. Mary's Hospital and treated him by continuous antiseptic inhalation. The carious teeth were extracted. He improved rapidly. When I examined him again on July 5 he had gained $3\frac{1}{2}$ lb. in weight, the dull areas were smaller, and the signs of active lesion had subsided. But his conditions of life are most unfavourable. On October 5 his weight was still 3 st. 10 lb., as in July, his cough had returned, and I found evidence of fresh implication of the left lung, the right lung being no more involved. Dr. Willcox kindly again admitted him into hospital, and he has once more improved and again gained weight. But one cannot but fear that the domestic conditions will prove too hard for him.

(29) Mr. N., aged 28, sent by Dr. Wilson, of Boston, May 7, 1909. Cough for six weeks; loss of weight, $2\frac{1}{2}$ lb. His sister, aged 25, died from phthisis after an illness of two years. Signs at all four apices and two lower areas, with some evidence of cavitation at the right apex. Gain of weight after three weeks of continuous antiseptic inhalation, 7 lb. A month later, after a stay at Margate, where he did not feel well, he was found to have lost 3 lb. The physical signs were stationary, but he had brought up some blood. This was due to bleeding gums round some very foul teeth. These teeth were extracted, and he again improved. On October 15 he was found to have gained 14 lb. since his first visit; there was no sign of activity in his dull areas of lung. He felt and looked very well, and was doing his full work without fatigue. He was dismissed as cured, but advised to wear his inhaler for two hours daily during the next three months.

(30) Mr. R., aged 30, seen at his home in East Ham, with Dr. McKettrick, July 27, 1909. Cough for two months; hæmoptysis, lasting several days, two weeks ago. Sputum mucoid. Extensive signs of tubercle in both lungs, at all four apices and five other areas, with some signs of cavity at the right upper apex and moist crackles at the left lower. I formed a very unfavourable opinion of his prospects. We at once instituted treatment by continuous antiseptic inhalation. On October 16 Dr. McKettrick wrote to me that "Mr. R. is now in Devonshire, putting on flesh rapidly, and his lung condition has improved very much."¹

This is, I think, a complete list of all the cases of incipient or early pulmonary tuberculosis which I have treated by continuous antiseptic inhalation, and in which I have been able to watch the progress or to ascertain the result. Every one of these thirty cases has shown marked improvement. This is surely a most encouraging fact. I consider twenty-two out of the thirty as certainly cured. In thirteen of them the cure has stood the test of time, for they have all remained well and in active work for a whole year. These are Cases 2, 3, 4, 5, 6, 7, 8, 9, 12, 13, 14, 15, and 16. To these may probably be added Case 1. Eight other cases, Nos. 18, 19, 21, 22, 24, 25, 26, and 29, are apparently quite well, but they have not yet stood the test of time. Five cases—Nos. 10, 11, 17, 20, and 27—I look upon as nearly cured. Only three cases remain about which one feels any doubt as to the favourable issue; they are Nos. 23, 28, and 30, and all these three cases have improved very considerably.

In the later stages of pulmonary tuberculosis the treatment by antiseptic inhalation will sometimes relieve cough and diminish expectoration, but it cannot be expected to cure. Let no one make trial of this method in advanced phthisis and then report that he "has obtained negative results." Of course he will obtain negative results. When the whole house is burning the most strenuous efforts of the fire brigade may be powerless, yet at its beginning the fire might have been extinguished by a mere pailful of water. If we are to succeed in arresting pulmonary tuberculosis, we must attack it in its earliest stage. At present cases are too often allowed to drift into a serious and even into an irremediable condition simply because the practitioner has not learned how to

¹ P.S., November 30, 1909. Mr. R. wore his inhaler day and night persistently for a month. For eleven weeks he has lived in the open air at Lyme Regis. He has gained 12 lb. in weight, has hardly any cough and almost no expectoration, and has been able to climb the Dorset hills. There is still a cavity at his right apex, with slight local tenderness and slight occasional local pain, but there is hardly any crepitant sound, and all his dull areas are much smaller. He feels well and hopes to resume work in January.

diagnose an early tuberculous infection by means of careful percussion. Yet this possibility is within the reach of all if a correct method be adopted. And here let me lay stress on the necessity for a complete relaxation of the patient's muscles. For the front of the chest, the patient must lie on his back on a comfortable couch and be placed completely at his ease. With the patient standing or sitting up it is impossible to ascertain the facts correctly, and some of the published pictures illustrating the "method of percussion," with the patient erect, are excellent examples of "how *not* to do it"! For the posterior aspect of the thorax, the patient should be sitting with his back to the physician; he should place his hands on the anterior aspect of the opposite shoulders, and should bend gently forwards.

In this paper I have endeavoured to maintain two propositions. The first is that the detection of pulmonary tuberculosis before any bacilli appear in the sputum is within the reach of any medical practitioner who is willing to take a little trouble. The second is that pulmonary tuberculosis detected at this stage can always be arrested by treatment by continuous antiseptic inhalation. It may be that some exception to both these statements may be found, but if these two propositions can be established, or if they are even approximately true, it is not too much to say that the practical extinction of the disease known as pulmonary phthisis is within our grasp. Since the essential point is a careful percussion, we may say that the secret of success is very literally *in our own hands*. I believe that within three or four years an enormous reduction of the mortality might be effected.

DISCUSSION.

Dr. WILLIAM EWART said it was a responsible task to express any opinion upon therapeutical conclusions largely based upon individual technique in observation. The earliest beginnings of pulmonary tuberculosis are exceedingly difficult to identify by physical signs. Both auscultation and percussion are valuable aids to diagnosis; but they are open, and in particular percussion, to many fallacies. Some of these are incidental to the finer percussion of the expert which can not only elicit a dullness, but can also manufacture it after the fashion of the manipulation of sound by the skilled instrumentalist. The chief risk from these artefacts is that they might at times be unconscious. But there are other fallacies bound up with the complex anatomy of the chest.

The normal difference in percussion between rib and intercostal space is not noticed in ordinary percussion; but when noticed in finer examinations it may be mistaken for a morbid sign. Again, there are fallacies connected with normal variations in the respiratory functions—as, for instance, those covered by the suggestive word “atelectasis,” when used in its attenuated clinical sense—to indicate a lack of full aeration of the lung or of some portion of it. Large-lunged people when at rest do not require to fill the whole of the lung with air, and in them an examiner might chance to find spots of relative dullness simulating disease. These are some of the risks of an attempt to diagnose early tubercle from percussion alone. Among many practical points for which the Section is indebted to Dr. Lees, he teaches us to relax the patient's muscles by the supine posture for the anterior examination. The same advantage might also be gained for the dorsal examination by the prone instead of the sitting posture, for so long as the spine is erect its muscles must come into play. These refinements are essential in any attempt at the early recognition of phthisis. The later stages of the disease do not present the same difficulties, and their diagnosis often is only too easy. With regard to *treatment*, in the absence of the full details of several cases for which there had not been time, he would ask Dr. Lees whether there were not other attendant circumstances in the cases which might have helped their improvement, or should the rapid gain in weight, which most of the patients showed, be attributed to the stimulating action on the mucous membrane? Some of the constituents used might act also as alteratives, and the turpentine would probably act upon the kidneys. Another important question was whether Dr. Lees believed that the inhalations, diluted with air as they were, were sufficiently strong to reach the bacilli and to act upon them as bactericides.

Dr. J. GRAY DUNCANSON said the subject of Dr. Lees' lucid and instructive paper was one which must always demand the closest attention of the medical man. When he was house physician in 1893, every case of pulmonary tuberculosis in the wards under his charge was treated by continuous inhalations by means of an oro-nasal inhaler of the form which Dr. Lees described. The substances used were creasote, rectified spirit, and carbolic acid in various combinations, and certainly the results, on the whole, were satisfactory. Since then, in general practice, when he could get patients to wear them, he had ordered the use of such inhalers. There was another class of case frequently seen which caused anxiety, as the disease was so incipient and the physical signs so slight that nothing definite could be determined either by auscultation or percussion. There might be some bronchial catarrh, but scarcely any temperature. Such patients often would not give up work, and could not use continuous inhalation. Ammoniated tincture of quinine, given when there were feverish symptoms, with the continuous administration of creasote in capsules, was frequently effective, and he did not find gastric symptoms induced thereby; but, on the contrary, when, as so often happened, these were already present, they were usually relieved by the treatment.

The PRESIDENT (Professor Cushny) said he would be glad if Dr. Lees, in his reply, would give the Section his idea as to whether the action of his prescription was an antiseptic one. The title of the paper suggested that it was antiseptic, but it seemed difficult to imagine that those traces of carbolic acid should not injure the healthy lung if they injured the tubercle bacillus.

Dr. LEES, in reply, thanked Dr. Ewart and Dr. Duncanson for their remarks, the latter especially for relating his interesting experience with an inhaler. He suggested that where there was a difficulty in getting the patient to wear an inhaler continuously he should wear it at night only; that could be easily done, without much discomfort, and it was beneficial, though, of course, not so good as what he had advocated. He declined to be drawn into an expression of opinion as to how the inhaler worked. It was certain that pulmonary tuberculosis was a microbic disease, and there was evidence that that disease was arrested by the use of a certain clinical method. Anyone could form whatever theories he liked as to the *modus operandi*.

The Action of some Diuretics.

By H. H. DALE, M.D., and P. P. LAIDLAW, B.C.

OUR object in this communication is to discuss the action of two substances which, in very different ways and in widely different connexions, have been described as specific diuretics, and both of which have recently been the subject of experiment by us in the laboratory.¹ It may seem almost paradoxical to deal thus in a single paper with the action of the drug known as Apocynum and that of the posterior lobe of the pituitary body. We hope to show some justification for this association.

The drug known to the United States pharmacopoeia as Apocynum has undergone various vicissitudes of enthusiastic recommendation, and almost equally enthusiastic disparagement. This, we imagine, is no uncommon history for a drug, which usually finds its proper level of appreciation at a point remote alike from those assigned to it by its earliest advocates and adversaries. The earliest claim which we have discovered for the introduction of this drug into civilized medicine is put forward on behalf of a certain Judge Gray, of Chautauqua county, who in 1820 learned from a tribe of Indians the medicinal virtues of the rhizome of *Apocynum cannabinum* in dropsy, and acquired a local reputation for the treatment of such conditions, handing down his information to two generations of medical descendants. The earliest descriptions of its use in medical literature are those of Knapp and Griscom, in 1826 and 1833 respectively. These observers subjected the drug to a laborious chemical examination, which, however, as might be expected from the state of organic chemistry in their day, was barren of result. These earlier observers mention diuresis as an effect of the drug, but their attention seems to have been drawn more particularly to its emetic, cathartic, and diaphoretic actions. Though the use of the drug seems to have spread in the United States, through the middle portion of last century, chiefly as a remedy for dropsy, great uncertainty seems to have prevailed as to the nature of its action. An Eclectic Dispensatory of 1855 recommends it in dropsy on account of its diuretic and hydragogue

¹ For details of the observations discussed and references to the papers cited, see Dale and Laidlaw, *Heart*, Lond., 1909, i, p. 138; and Dale, *Biochem. Journ.*, Liverpool, 1909, iv, p. 427.

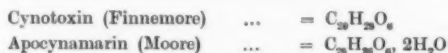
cathartic action: the latter action is emphasized in several publications as the basis of its remedial effect in ascites, while its emetic action in large doses is frequently mentioned. The accounts of this period leave the reader merely with a general impression that the drug probably contains a principle belonging to the group of the drastic purgatives. In 1876 a pharmacological examination by Husemann showed that it has the action of a cardiac tonic of the digitalis group. In 1883 a chemical and pharmacological examination was carried out in Schmiedeberg's laboratory and two preparations obtained—one soluble in alcohol, almost insoluble in water, to which the name Apocynin was given; the other a water-soluble, saponin-like body which was named Apocynein. Of these the former, apocynin, was especially toxic, producing systolic arrest of the frog's heart in very small doses. Unfortunately, neither of these products of Schmiedeberg was crystalline or had any of the characteristics of a pure substance. This has indirectly been a factor in the discredit of apocynum, since the manufacturers, working on a large scale, have been preparing what is doubtless a highly purified version of Schmiedeberg's active principle, and selling, as apocynin, a beautifully crystalline substance practically devoid of activity of any kind. Murray, in 1889, reported the beneficial action of the tincture of apocynum in cases of cardiac dropsy, the pulse being regularized and reduced in rate, the volume of urine increased. It is noteworthy that he used it with success in cases in which digitalis had to be abandoned on account of its emetic action. Murray quotes unpublished pharmacological experiments by Rose Bradford, who found that the tincture of apocynum acted almost entirely on the heart, the beat of which was strengthened, but was simultaneously so reduced in rate by vagus inhibition that the blood-pressure did not rise. According to Bradford the drug had practically no vaso-constrictor action. A year previously, however, Sokoloff had examined its physiological action very thoroughly, and his description gives a good picture of the various stages of an action characteristic of the digitalis series. According to Sokoloff the preliminary stage of vagus inhibition gives way to a very great rise of blood-pressure, due to powerful and very rapid heart-beat and intense vaso-constriction, the action ending in sudden heart-failure. An examination by H. C. Wood, Jun., in 1900, added but little to Sokoloff's description; he rightly, however, ascribed to direct peripheral action the vaso-constriction which Sokoloff had attributed chiefly to action on the vaso-motor centre. Wood also made the important observation that the commercial crystalline apocynin was almost devoid

of activity, and that an apocynin which he prepared himself, though active when first prepared, became less and less so with successive purifications by recrystallization.

Meanwhile some revival of interest in the clinical use of the drug had occurred. A communication on its use made by Woodhull in 1897 produced quite a crop of reports from physicians, who claimed priority in, or independent discovery of its use, or were interested on one side or the other in the controversy concerning its value. Prominent among these was Dabney, who appears to have learned of the drug independently from native sources, and was so convinced of its efficacy that he asserted, in his own words, "an absolute confidence in my ability to entirely discard the use of the trocar whenever a tincture, fluid extract, or infusion of the bark of the root of the genuine *Apocynum cannabinum* can be obtained." Dabney introduced a new element of confusion and uncertainty into the subject by his insistence on the necessity of insuring that the drug was of this one species. In his own words: "Manufacturing chemists, through ignorance, carelessness, or greed, have foisted upon the profession a number of radically different drugs under the name of *Apocynum cannabinum*; hence results obtained by different physicians using different medicines under a common name varied so widely that the drug was eventually discarded altogether. The *Apocynum androsæmifolium*, an utterly worthless weed, is frequently substituted for the *Apocynum cannabinum*, which it closely resembles in appearance." There does not seem to have been any experimental justification for this conclusion; on the other hand, it obviously furnished an enthusiastic advocate with a ready means of explaining away any untoward or unsuccessful result reported with apocynum, as having been due to failure to obtain the right species.

Our object in entering at this somewhat tedious length into the chequered history of this drug is to furnish an adequate explanation for its anomalous position at the present day. When we find the drug vaunted by some as an unfailing specific diuretic in dropsy, being, according to one writer, even in hydrocephalus, "the only remedy on which we can depend"; by others admitted as a remedy, but regarded as "a mere evacuant of dropsical effusions . . . possessing this value in common with jalap and other hydragogue cathartics"; by others, again, admitted to have a digitalis action, but discarded on account of its emetic and cathartic properties; recommended as a diaphoretic and expectorant, a tonic and alterative, as a remedy for intestinal worms, as a cure for mild cases of intermittent fever, and even as a local application

and internal remedy for rattlesnake bite: when we read, on the one hand, that one species alone is active, and yet find the United States Pharmacopeia admitting others under its official definition of apocynum, it is hardly surprising that the great reputation which the drug has enjoyed locally and at times has not crossed the Atlantic. Nevertheless, it would be a matter for regret if these uncertainties and contradictions should lead to the final disuse of a drug in favour of which there is such an accumulation of testimony, even if one may suspect that this is tinged with enthusiasm. Nothing could be more conducive to a juster appreciation of such a drug than a thorough chemical examination, leading to the identification of its active principle. It was particularly fortunate, from one point of view, that it attracted almost simultaneously the attention of the Pharmaceutical Department at Guy's Hospital and of the Wellcome Chemical Research Laboratories, and that while Mr. Finnemore was investigating *Apocynum cannabinum* at the former, Dr. Moore was at work in the latter on a quantity of the drug which was botanically identified as being *Apocynum androsæmifolium*. Mr. Finnemore isolated the substance which has been for some years obtainable as apocynin, identified it as acetovanillone, and confirmed his identification by synthesis. Dr. Moore has obtained the same substance from *Apocynum androsæmifolium* together with its glucoside, which has proved to be no more active than the acetovanillone itself. Finnemore and Moore each isolated also from the species which he examined a crystalline neutral principle, not glucosidal in nature, this principle being found in each case by one of us to have a very powerful action of the digitalis type. So far a good deal of uncertainty was removed. The suggested inactivity of *Apocynum androsæmifolium*, at least as regards this most characteristic action, was shown to be wholly mythical, and we had the opportunity of investigating the action of a crystalline principle, obtained without difficulty in a state of chemical purity, instead of extracts of unknown, probably varying, and certainly not uncomplicated action. It should be mentioned that there is no certainty as yet, from the chemical point of view, that the substances obtained by Finnemore and Moore are the same, though there is every likelihood of their being so. They were named differently and formulated differently by their discoverers:—



We found, however, at an early stage of our respective investigations that we were dealing with substances which were at least physiologically

indistinguishable. We therefore joined forces and finished the investigation together. Without taking part in a chemical discussion, which is not our concern, we cannot adopt one name or the other; but we have used Finnemore's cynotoxin and Moore's apocynamarin with absolute indifference, so that, apart from the labels on our records, we should not know which was employed in any particular experiment. The action is very similar to that of the other members of the digitalis group. If the substance enters the circulation slowly, as by hypodermic injection, or, still more so, by absorption from the stomach, there is an initial stage of very marked retardation of the heart-beat by vagus inhibition. In a cat with a normal heart-rate of about 200 per minute, to which a dose of 2 mg. was given by the mouth twice daily for eight days, the beat was kept down to an average rate of about 140 per minute, sinking to 110 per minute, or even lower, in the periods up to three hours after each administration during which the animal might be considered to be under the full effect of the dose. Vomiting occurred only once during this period, and was clearly not due to irritation of the gastric mucous membrane, for the stomach of the animal, which was killed after the experiment was complete, was perfectly normal. When fully under the drug the animal exhibited distinct drowsiness, an effect which some observers have attributed to extracts of apocynum. It is, of course, difficult to be certain of the genuineness of this effect. An animal of nocturnal habit, like the cat, so readily falls into a drowsy condition in the daytime, when left undisturbed in conditions of warmth and comfort, that it is almost impossible to recognize a mildly sedative as distinguished from a definitely narcotic effect. Comparison with other animals, however, and with the same animal in the intervals during which the action of the drug had weakened, inclined us to the conclusion that such a sedative action was actually present. If so, the point may be of practical service in the treatment of heart disease, where the breakdown in compensation is associated with restlessness and insomnia. During the whole of this experiment the cat remained in good condition, ate readily, and put on weight rapidly. In fig. 1 the rates of the heart-beat during the experiment are plotted. It should be noted that the omission of the second dose on one day (the third) was followed by recovery of the heart-beat to near its original rate on the following morning; also that after the last administration the effect of the drug rapidly passed off. There is no evidence, therefore, of anything like cumulative effect with this rate and method of administration.

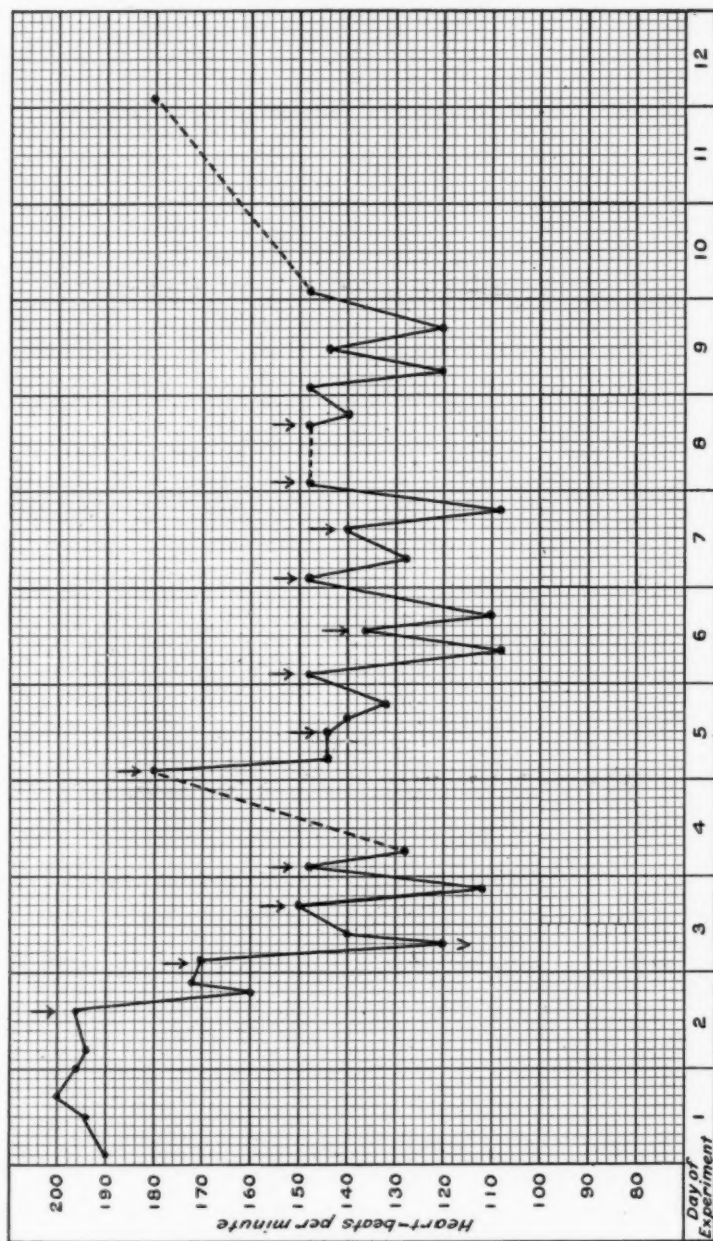


FIG. 1.

Experiment on Cat. ↓ = Administration of 2 mg. apocynanarin by the mouth; V = vomiting.

The effects on the heart-beat are illustrated in a more acute form by subcutaneous injection of a small dose. Fig. 2 shows the result of injecting 1 mg. hypodermically into a cat. It will be seen that the rate of the heart-beat falls rapidly, and the stage of excessive inhibition, with

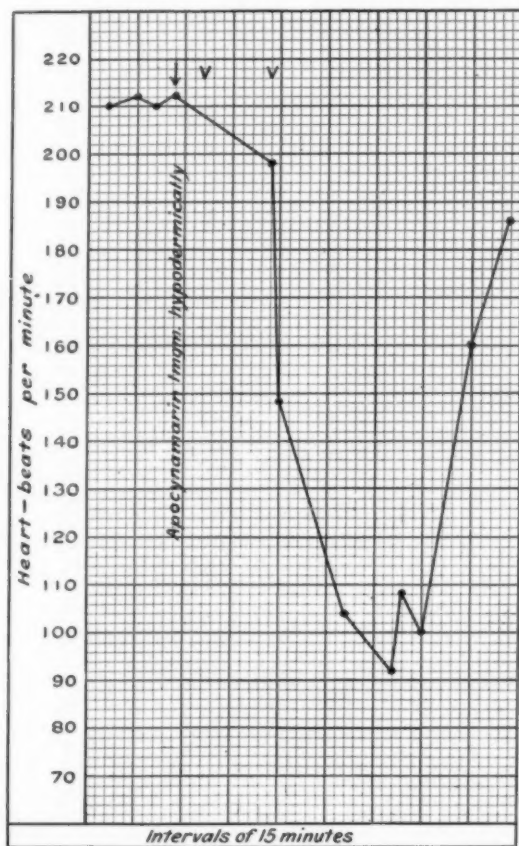


FIG. 2.

Experiment on Cat. ↓ = Hypodermic injection; V = vomiting.

irregular interruptions of the heart-beat, is soon reached. Recovery is equally rapid, and the whole effect is practically over in one hour. It should be noted that vomiting is readily caused by this method of

administration—a further indication that this effect is not the result of gastric irritation, but must be attributed to the action of the drug, after absorption, either on the vomiting centre or directly on the musculature of the stomach.

Fig. 3 illustrates the action on the pulse of a monkey. An excessive vagus inhibition occurs as before. In this case, however, the third stage

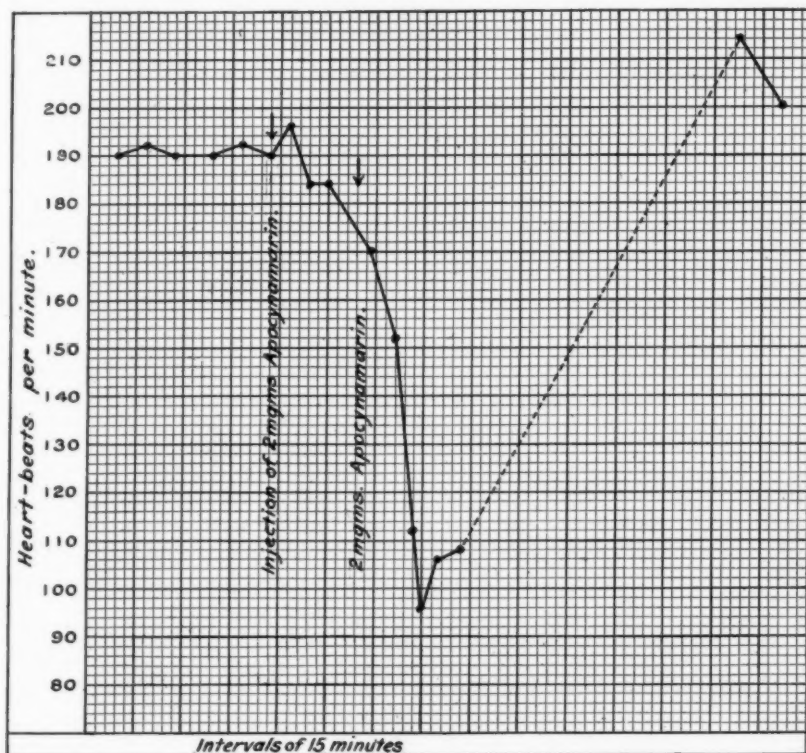


FIG. 3.

Experiment on Monkey. ↓ = Hypodermic injection.

of digitalis action—escape from vagus inhibition, with very rapid pulse—is produced in typical form. From this, however, the animal recovered after a few hours. The effects of the drug can be analyzed more closely by mechanical records of its action on the arteries, heart, and viscera of

the anæsthetized animal, or on isolated organs. It is then seen to have a powerful action on the heart, comparable to that of strophanthus, combined with the peripheral excitation of marked tonus in plain muscular organs, including the arteries, the spleen, the uterus, the alimentary canal, and the urinary bladder. On voluntary muscle it has no action. (Tracings illustrating these actions were exhibited by the epidiascope.)

In its action as a diuretic we have not found that apocynamarin (or cynotoxin) occupies an exceptional position in the digitalis series. As others have found with other drugs of this group, the diuresis, accompanied by expansion of the kidney, is much more easy to demonstrate on the rabbit than on the cat or dog. In the latter animals the first effect of an intravenous injection is to cause diminution of the kidney volume and diminished diuresis; this gives way to a secondary expansion of the organ and acceleration of the flow of urine as the blood-pressure falls. The diuresis is best obtained in the cat by injecting the drug hypodermically and allowing it to be absorbed slowly. One then obtains during the earlier stage of the action, in which the rise of blood-pressure due to vaso-constriction is held in check by vagus inhibition, a quite notable increase in the rate of the diuresis. This is a fact which is hard to reconcile with the generally received idea that the diuresis produced by digitalis and its allies is due to increased cardiac activity, for we have here a case of vaso-constriction accompanied by diminished heart-output per unit period sufficient to prevent the rise of arterial pressure which the vaso-constriction would otherwise produce; nevertheless, there is diuresis. On the other hand, perfusion and other experiments have shown that the renal arteries are not immune to the general vaso-constrictor action of these drugs.

It may be suggested that two factors are concerned: (1) A relative insensitiveness of the renal arterioles, leading to a redistribution of the blood in the arterial system of such a kind that more flows through the kidney at the expense of other organs even when the general arterial pressure remains constant; and (2) a fall of venous pressure, owing to accumulation of blood on the arterial side: the renal veins, being near the heart, would be especially affected. These combined influences will lead to increased rate of circulation through the kidney, even, as Loewi has shown, when the organ is prevented from expanding by embedding it in a mass of plaster of Paris. Another point, which is worth attention in this connexion, is the apparent association in the digitalis series of a powerful action on the peripheral arteries with diuretic action.

Thus the two substances which seem to have the most powerful vaso-constrictor action among the members of this series—squill and apocynum—have also a special reputation as potent diuretics. Whether this is to be taken as indicating an actual bio-chemical affinity between the plain-muscle cell and the renal epithelium, so that the latter is directly irritated by substances which excite the former to tonus, is a point on which we are not prepared to offer evidence. It is possible that the diversion of blood to the kidney owing to the prevalence of vaso-constriction in other organs, as above suggested, is adequate to account for this association between vaso-constrictor and diuretic action. It is dangerous also to lay too much stress on the clinical reputation of a drug; it must not be forgotten that digitalis itself ranked as a specific diuretic before its action on the heart was appreciated.

Whatever be the explanation of the diuresis produced in the normal animal, there is not much room for doubt that a principal factor in the restoration of this function, in cases of anasarca of cardiac origin, is the strengthening and regulation of the heart-beat itself, with the consequent relief of the venous engorgement. One other observation which we made incidentally is worth mention, though neither original nor unexpected. The diuretic effect obtained in the rabbit by injecting apocynamarin (or cynotoxin) is very much greater if the injection is made subsequent to a previous dose of one of the purin diuretics (such as caffeine, or theophylline), the visible effect of which has ceased. A similar result was obtained by Jonescu and Loewi, who found that in the dog, after strophanthin, a pronounced diuresis was produced by a dose of caffeine too small to have any such effect when given by itself. The general vaso-constriction and improvement of the heart-beat induced by strophanthin, or the local effect of the caffeine on the kidney, while independently inadequate to produce a sufficient diversion of the blood-stream through the kidney to evoke a perceptible diuresis, acting together became efficient. This reinforcement of digitalis diuresis by xanthin-base diuresis is not new to practical medicine, but the laboratory demonstration of it was so striking as to lead us to investigate the possibility that some other constituent of apocynum might have such a reinforcing effect on the diuretic effect of apocynamarin. We have found no evidence for any such action, and our present view is that the whole activity of the drug in this respect is represented by this active principle. If this view be correct, apocynum must be removed from the exceptional position allotted to it by some clinical advocates and be placed alongside the other members of the digitalis series. It is

satisfactory, on the other hand, to think that the isolation of the pure principle will at length enable it to be tried clinically without any lingering doubts as to the correctness of the species employed or the activity of the preparation. From some points of view we believe it should have advantage over some members of the series. In our experiments on administration by the mouth the effect on the heart was rapidly attained, and could be maintained without any sign of tolerance on the one hand or cumulation on the other. The comparative ease of isolation and purification and the stability of the active principle is another point which should undoubtedly be in its favour, when one considers the difficulty of obtaining in commerce the active principles of digitalis and strophanthus in anything approaching uniformity. On the other hand, the cathartic action of apocynum will probably not be obtained with the pure principle. This, however, we are disposed to regard as an advantage, for some other cathartic can be given if desirable, while it is well not to be hampered or limited in administering a cardiac tonic by its concomitant action on the alimentary canal.

The other diuretic action to which we propose to refer is that of extracts from the infundibular lobe of the pituitary body. The action of extracts of this organ in producing a rise of blood-pressure was discovered by Oliver and Schäfer in 1894. Howell showed that the action was limited to the posterior lobe, developmentally related to the central nervous system. The analogy to the active principle of the suprarenal gland, limited to the medulla, which in development is related to the sympathetic system, was clear, and was indicated by Schäfer and Herring. The latter observers, in amplification of an observation by Schäfer and Magnus, studied the marked diuretic action of the extract and concluded that this was produced by a specific renal excitant, quite distinct from the principle acting on the blood-pressure.

In examining the action of the extract on various organs and tissues we found that it had a general action on plain muscle very similar to that of certain members of the digitalis series. This action has no apparent relation to innervation by any division of the autonomic system; arteries such as the pulmonary and coronary branches, which receive little or no vaso-motor nerve supply, are stimulated to constriction equally with those governed by sympathetic nerves; the uterus of the cat, which relaxes in the non-pregnant condition and contracts when pregnant, in response to sympathetic nerve impulses or suprarenal extract, contracts in any condition in response to infundibular extract. Similarly, with other plain muscular organs and systems, the response

to the pituitary principle is invariably contraction. This action is not affected by ergotoxin, which eliminates the motor action of sympathetic nerves or suprarenal preparations. So far, then, the pharmacological affinities of this extract are with the digitalis series. Like the digitalis drugs, again, it has practically no action on voluntary muscle. On cardiac muscle its action is comparatively slight. It produces a strengthening of the beat, accompanied by some degree of retardation, but the effects are small. Nothing comparable to the excessive excitation and final systolic rigor produced by the digitalis drugs is seen. If the experiment is made on a heart perfused under constant pressure the initial augmentation of the beat gives way to a secondary weakening. This, however, is doubtless partly attributable to the constriction of the coronary arterioles. In the entire animal, when this constriction is antagonized by the great general rise of systolic pressure, the augmentation of the heart-beat, accompanied by retardation, persists for a long time.

This similarity in its action on the circulatory system to the digitalis group suggested a closer examination of the evidence which led Schäfer and Herring to attribute the diuretic effect to a separate principle. This may be classed under two headings:—

(1) The dissociation of the pressor effect from the diuretic effect in time-relations and relative prominence.

(2) The alleged selective destruction of the pressor principle by certain enzymes and chemical agents.

The dissociation in time-relations of the two actions is seen to be similar to that with which we are familiar in the case of the digitalis drugs. The rise of blood-pressure is attended by initial retardation or cessation of diuresis, followed by increased diuresis, which attains its maximum after that of the arterial pressure, and lasts long after the pressure has returned to the normal. A closely similar sequence may be observed, as we have shown, with apocynamarin or strophanthin, which certainly do not contain separate pressor and diuretic principles. The fact that a second injection will frequently produce a weaker but distinct renewal of diuretic action, without producing an obvious rise of blood-pressure, is also conceivable on the view that one principle produces both effects. The blood-pressure effect is more complex: a heart-depressant principle exists alongside the pressor principle in the extract, and the action of this becomes more significant as the pressor effect becomes weaker with repeated injections. It may be doubted, also, whether effects can be properly compared which depend for their recognition on methods of

such different sensitiveness as the manometric record of blood-pressure and the drop record of diuresis. We have observed cases in which contraction of the uterus was produced by a second injection, which caused practically no rise of blood-pressure; but this would not justify the conclusion that the principle acting on the plain muscle of the uterus is distinct from that which causes vaso-constriction.

We have not been able to find any evidence for a selective destruction of one action or the other by ferments. In our experience both actions are equally unaffected by pepsin, equally readily destroyed by trypsin, and both slowly destroyed at about the same rate by oxidation with hydrogen peroxide. We are disposed, therefore, to regard the evidence brought forward in favour of the existence of two principles as inadequate.

It appears to us that the evidence points to the diuretic action of pituitary extracts as being of a similar type to that produced by the digitalis group of drugs. In the case of pituitary extract, again, the association of vaso-constrictor action, in this case accompanied by but slight action on the heart, with a powerful diuretic action is very marked. It is worthy of mention that we have observed with large doses the production of renal hæmorrhage, as some have recorded after large doses of squill.

The PRESIDENT (Professor Cushny) said he had listened to the paper with much interest and agreed with it all. He had tested the cruder forms of apocynum, but not those such as Dr. Dale had been using. One of the tracings exhibited, showing curious rhythmic irregularities, was of especial interest, and he would be glad of any explanation of the phenomenon. The purgative effect of these substances gave rise to difficulties which he trusted might in the future be got rid of. All the drugs, such as digitalis and strophanthus, acted on the alimentary tract, and when experimenting with euonymin and helleborein he always found purgation before marked heart effects could be obtained.

Therapeutical and Pharmacological Section.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

December 7, 1909.

The Therapeutical Value of the Lactic-acid Bacillus.

A DISCUSSION.

Opened by GEORGE HERSCHELL, M.D.

THE subject which is to be discussed to-night is of considerable importance, because there is more than a slight danger of a valuable therapeutic agent being brought into unmerited disrepute from its indiscriminate use in unsuitable cases. It is one of the most unfortunate features of the age of competition in which we live that no sooner is some method of treatment devised which may possibly be of real use than manufacturing chemists hasten to place the material used upon the market. This of itself would be a convenience if they would stop there ; but unfortunately they go further, and attempt to create a demand for it, usually long before its clinical trials have been finished and its therapeutic use placed upon a firm basis. This they effect by compiling a pamphlet containing any suggestion for its use in diseased conditions which they may find in the writings upon the subject. Moreover, the conditions which are supposed to be benefited are so loosely described, and definite indications so invariably omitted, that the drug becomes widely used in conditions in which it cannot possibly do any good. Failure naturally ensues, and the use of what may be a really useful agent in proper cases is discredited. It has been so with the lactic-acid ferments. Not only have manufacturing chemists placed

numerous preparations upon the market, but even the milkmen have entered into the arena and have inundated medical men with particulars of soured milks manufactured by them, and have also been kind enough to attempt to teach them their use by means of papers of clinical indications.

As an illustration of my statements I have before me the circular of a firm of milk purveyors which suggests the use of lactic-acid bacteria in the treatment of dyspepsia, entero-colitis, gastric ulcer, rheumatism, &c. The text of the circular commences with the statement that "The great value of lactic-acid organisms in the treatment of certain diseases, especially in the case of indigestion accompanied by flatulence, is conceded now on all hands." It is needless in an assembly like this to point out how utterly useless and misleading is this assertion. We are all perfectly familiar with the fact that indigestion accompanied with flatulence is very often a pure neurosis, or may be due to atony of the stomach, gastroptosis, or hyperchlorhydria. It may connote many and diverse conditions. We may have a neurosis with swallowed air, atony with regurgitation of the pancreatic juice into the stomach with evolution of carbonic-acid gas from the decomposition of its carbonates, or retention and decomposition of food residues from several causes or hyperchlorhydria. In any of these it is difficult to see in what manner lactic-acid bacilli could act beneficially. Again, the fermentation may be intestinal in the small intestine with regurgitation of the gases produced, the process being one which produces lactic acid, butyric acid, and various gases. Here the probability is that the introduction of lactic acid or lactic-acid producers will aggravate the existing evil. In entero-colitis, as I shall presently show, the use of lactic-acid bacilli must be restricted to certain definite forms. In gastric ulcer there are, as far as I am aware, no clinical records of their successful use, but, as we carefully eliminate acids from the diet of such patients, there is no reason why an exception should be made in favour of the Bulgarian bacillus. In rheumatism, if you accept the old hypothesis of Prout, Todd, and Richardson, that the causative agent is an excess of lactic acid in the system, you must logically reject lactic-acid therapy. If you incline to the more recent theories, such as the one that rheumatism may be caused by the absorption of the discharge of a pyorrhœa alveolaris, you must do the same, as there is every probability that the organism which has such a large share in producing both pyorrhœa and dental caries—namely, the buccalis maximus—is identical with the Bulgarian bacillus;

in fact, it is specifically stated by Heinemann and Hefferan¹ that such is the case.

We see, then, that the instructions for use as supplied by the dairyman are at least misleading and liable to cause the use of the Bulgarian bacillus in unsuitable cases, and the same may be said for most of the literature issued by the manufacturing chemists. But, even if used upon suitable cases, the scientific value of the results obtained will depend entirely as to whether the observer has satisfied himself that the preparation which he is using contains living Bulgarian bacilli. If you are not sure that a certain tablet contains the Bulgarian bacillus, any cure obtained with it will only prove logically that *this particular tablet* is a useful agent in the disease in question, but is no proof of the usefulness of the Bulgarian bacillus. And, as I have proved by personal examination (and others more skilful than myself have done the same) that not more than two or three out of the dozen or more preparations upon the market contain any Bulgarian bacillus at all, it must be obvious that the published clinical records must be accepted with great reservation. As I have already expressed my views in print upon the use of commercial soured milks, I shall now confine myself to the consideration of the preparations in tablet form. If properly made, and containing living Bulgarian bacilli, this should be the ideal method of using lactic-acid therapy in every-day practice, the use of liquid pure cultures being reserved for serious and acute cases. The reason is that, although it is unavoidable that the bacteria of the atmosphere fall into the substance from which the tablets are made, they are unable to develop, as they find only dry powders which are not a suitable medium for their growth. When swallowed, the lactic-acid bacilli outgrow them and inhibit their further development. The makers' assertion as to the contents of the tablets cannot, unfortunately, be taken for a guarantee, as anyone can prove to his own satisfaction that the majority of tablets contain only, as a rule, the Hueppe and some yeast. If any Bulgarian bacilli are present, they are too few to be of any practical use. But in the majority of cases the inference must be made that the makers have not put them into the tablets at all, but have contented themselves with the weaker lactic-acid bacilli, which, although they curdle milk, are destroyed in the stomach and will not produce the effects which are claimed for the Bulgarian bacillus.

¹ "A Study of *Bacillus bulgaricus*," *Journ. of Infect. Dis.*, Chicago, 1909, vi, p. 305.

As a climax, we have a chocolate cream placed upon the market by manufacturers who, apparently accepting as gospel the yet unproved hypotheses of Metchnikoff, invite a credulous public to consume them daily in the vain hope that they may extend their allotted span of existence. The sample which I examined contained no cream and no Bulgarian bacillus. Bacteriological investigation revealed a few Hueppe, a large quantity of yeast, and some large putrefactive organisms. The testing of any tablet is quite easy and quite within the power of any medical man, as it involves merely dropping the material to be examined into a test-tube of litmus milk and keeping it warm for a few hours in a thermos flask filled with hot water. After that, a film preparation can be made and stained with Gram and counterstained with neutral red. I show you here two tubes of litmus milk in which have been cultivated two different varieties of tablet. In this one the curd is still good and firm, and the litmus has been reddened by the lactic acid produced. In the other you will find, microscopically, all kinds of filth. The tablet used in each case was one having a large sale.

In all serious and acute cases in which we wish to secure the action of the Bulgarian bacillus as quickly as possible, we should make use of a pure liquid culture prepared in a laboratory by methods which have been devised to secure the largest growth, microscopically examined against contamination before delivery to the patient, and used within a few days of making. Such a culture will keep good in the cold for about a week. One of the best nutrient mediums on which to grow the culture is a weak solution of malt extract, with the addition of a small quantity of peptone and common salt.

Having secured a preparation of the Bulgarian bacillus free from contamination and really containing living bacilli, we may expect to obtain good results in the following groups of cases:—

(1) Cases depending upon abnormal putrefaction of proteids in the intestinal tract. This group will cover a wide field and account for a great many of the affections met with in daily practice. The effects produced may depend (a) upon local irritation of the products of the putrefactive process.—Under this heading we find acute enteritis and entero-colitis; chronic colitis; some forms of diarrhoea, especially in children; mucous colitis and muco-membranous colitis. (b) Auto-intoxication with the products of putrefaction.—In this group we find many cases of gradual failure of health, especially in elderly people; some cases of skin disease, many cases of neuræsthenia, malnutrition

in children, some cases of anæmia, and quite possibly some cases of arthritis, neuritis, and other obscure affections of nerve and muscle.

(2) Some forms of constipation.—If lactic-acid bacilli are administered indiscriminately in all cases of constipation, you will have many more failures than successes. These agents obviously cannot restore power to an atonic muscle nor sensibility to an exhausted nerve. But exceedingly good results will often be obtained in those cases in which the constipation depends upon a defective production of the gases and acids which are the natural excitors of the intestinal peristalsis. These cases can usually be recognized by the diminution in the solid material of the fæces as compared with healthy stools whilst the patient is on an intestinal test diet.

(3) Lactic-acid bacilli should theoretically be of use to the operating surgeon, who, by administering a pure culture for a few days before an operation upon the intestine, can certainly render its contents much less toxic.

Before concluding my remarks this afternoon I wish to draw attention to a point of great importance, the neglect of which is, I am sure, the cause of many failures in the use of lactic-acid bacilli. It cannot be too distinctly impressed upon our minds that there are two distinct morbid processes which may take place in the intestine:—

(1) A putrefaction of proteids, due to the multiplication of proteolytic organisms; and

(2) An abnormal fermentation of carbohydrates, from the excessive growth of saccharolytes.

It is the first of these only which we can hope to influence by the use of the lactic-acid bacilli, and in practice it is essential that we diagnose the exact condition before attempting anything in the way of treatment. The abnormal fermentation of carbohydrates, so familiar to all of us as the intestinal indigestion of starch, can only be increased by the introduction of lactic-acid bacilli which produce the same kind of fermentation with the same products. The method of distinguishing the two conditions is not difficult and quite within the range of daily practice, depending merely upon an examination of the stools. The tests are as follows:—(a) The reaction of the stools: putrefactive stools are alkaline; fermenting stools are acid. There are, of course, some exceptional circumstances in which the reaction is not clear; but under these we make use of (b) Strasburger's fermentation apparatus, by which we can find out the kind of process to which the fæces are predisposed,

and thus determine which process is probably taking place in the intestine. About 5 grm. of fæces are placed in the lower receptacle and the tube directly above it filled with water. The whole apparatus is then kept at 37° F. for about twenty-four hours. The amount of gas formed is estimated by the quantity of water forced into the third tube. If over a third of this tube is filled with water, and if the reaction of the fæces after the operation is distinctly acid, then we can assume that the decomposition in the intestines is a fermentation of carbohydrates and the administration of lactic-acid bacilli is contra-indicated. (c) The examination of Gram-stained cover-glass preparations counter-stained with neutral red. In healthy stools the Gram-negative organisms will preponderate, the colon bacillus being Gram negative. In abnormal putrefaction, in proportion as the aerobic bacteria are replaced by strict anaerobes, most of which are Gram positive, so the blue-stained organisms are in excess. As a general rule, to which, however, there are many exceptions, we may say that, if we find the stools predominantly Gram positive, lactic-acid bacilli are indicated and will probably do good. If, on the contrary, we find the majority of the bacterial flora Gram negative, we shall probably do more harm than good by attempting to acclimatize the Bulgarian bacillus in the intestine.

DISCUSSION.

Dr. GOODBODY said that Dr. Vaughan Harley and he had been working on the subject from the chemical point of view, and their results were very much in agreement. There was great difficulty in determining by chemical means how much the decomposition in the intestines was influenced by the Bulgarian bacillus. There was a certain diminution in the amount of aromatic sulphates when one put a patient on a definite diet, examined him, and then gave the Bulgarian bacillus. But it was not by any means so constant as one would think it ought to be from the way the patients improved in suitable cases. There had always been found to be a diminution in the intensity of the qualitative reaction of urobilin in the fæces. There had been no exception to that. The only other point from the chemical side was that the absorption of nitrogen apparently tended to diminish while the lactic-acid bacillus was being given.

Dr. VAUGHAN HARLEY remarked that, as Dr. Goodbody had said, their chemical work on the subject was in the embryo stage; it was very difficult to get a suitable patient, keep him on a particular diet, and at the same time give lactic acid. Only in one case could they get a month's proper observation; several cases were watched for a week or a fortnight, but he did not consider they sufficed to form any judgment. In certain cases a distinct chemical change had been found. Fermentation of the feces with Strasburger's fermentation tube was the best means of making a diagnosis as to which case was suitable for the treatment. If the feces were acid, due to increased intestinal fermentation, one could say that the patient did not need the Bulgarian bacillus. If the feces were at first alkaline, but yielded a fair quantity of gas in the fermentation tube, showing an acid reaction after, this indicated intestinal fermentation, and he could not say that he had had any results in these cases. If, however, the fresh feces were alkaline and yielded no gas from the fermentation test, and also remained alkaline after twenty-four hours, showing that intestinal putrefaction was to the fore, good results were often obtained. When the lactic-acid bacillus was given there was an increased quantity of feces discharged, even when the patient was kept on the same diet; this increase was largely due to the larger quantity of water in the stools. In the case of the patient who was watched for a month, during the first week, both prior to and while taking the lactic-acid bacillus, the water in the stools was 73 per cent. and 75 per cent.; while during the last analysis, when he had had a month's treatment, it had increased to 82 per cent., though no medicines were being taken. In this case also there was a slight increase in the quantity of nitrogen in the stools while the lactic acid was being taken. The psychological factor could scarcely come in, as the patient was not educated, and did not realize that he was taking anything except medicine. There was no marked chemical evidence that it was valuable in all cases. There was much talk about it, and patients would often ask for it. No doubt, as Dr. Herschell said, the public were being instructed very wrongly about it, and they thought that all their diseases were curable by the method. A man recently told him (the speaker) that doctors would soon be a thing of the past, and that he was led to understand that no medical man would be required, because sour milk was all-sufficient. This man had disease of the heart, for which he was content to take sour milk. Many of the tablets sold were useless, and a kind of tablet which was useful at one time was not at another, so one never knew whether a particular result was psychological or not. One of the best cases of supposed recovery with the treatment which he knew was that of a man three years ago who had intestinal dyspepsia, probably due to taking too much champagne. He took the tablets and cured himself at once; consequently he said that all medicines were useless. He had seen that gentleman since; champagne was still to the fore and he had got all his old symptoms back, and

the tablets were not now doing him any good. One could only say that his former cure was probably chiefly psychological. And it was true that in the majority of cases where there were good results from sour milk, the patient had functional dyspepsia, and not any structural change. In slight dyspepsia, where there was increased intestinal putrefaction as against increased intestinal fermentation, he thought he had seen, even after the administration of rather doubtful tablets, distinct results. Perhaps these results were partly due to the tablets, but also to the fact that the diet contained sour milk, and patients under the treatment adhered more strictly to the diet than under ordinary circumstances. At present there was great danger of the public thinking too much of sour milk, and there was a liability of doctors doing the same; he had heard of doctors expecting results which to others seemed very unlikely. The subject required careful consideration, especially as it was so difficult to get pure cultures of Bulgarian bacillus. He had obtained extremely good results in a nursing home from milk containing yeast alone. The patient was under analysis, and there was a distinct diminution of urobilin in the stools. Mr. Goadby was good enough to make an examination of specimens of the sour milk, and they contained no Bulgarian bacilli; there were pure yeast and a few lactic-acid cocci.

Dr. BULLOCH said he wished to speak only on the bacteriological aspect, and especially on the examination of lactic-acid preparations on the market. During the last year Mr. J. E. Adler, working under his direction, had examined bacteriologically all the preparations of lactic bacilli which could be obtained. These included eleven dry preparations in the form of tabloids, nine milks, and one cheese. Without making any invidious comparisons, he had not found one preparation which contained *Bacillus bulgaricus* in a state of purity. Very many of the liquid preparations were grossly contaminated, and many of the dried preparations were either sterile or contained spores of bacteria other than those which produce lactic acid. He would like to hear from Dr. Herschell which solid preparation contained the lactic-acid bacillus (*Bacillus bulgaricus*) (Dr. Herschell said, "Trilactine"),¹ as it dies out relatively easily seeing that it does not spore. In some instances it was apparently not the intention of the manufacturer to supply pure cultures of Massol's bacillus, as he tried to mix all the bacteria grown by Massol in soured milk. In any case the majority of milks and milk preparations on the market were quite unreliable from a bacteriological point of view, and were no doubt in some instances made by those not skilled in bacteriological technique. More than one manufacturer

¹ Since the meeting of the Therapeutical Section another set of samples of "Trilactine" have been examined and have been found to be as variable as those on previous occasions. No two tubes gave the same type of fermentation. The number of bacteria was very variable, and while in some tabloids Massol's bacillus could be found, in others it could not. Its quantity, in any case, was trifling compared with the other bacteria present.

made reference to three papers, by Metchnikoff, which were said to contain the fundamental data of lactic therapy. These references were quite erroneous, for on looking them up he found that one paper referred to the question of senility in cockatoos, the other two being researches on syphilis. The researches on the chocolate-cream "massolettes" at the London Hospital had given results differing entirely from those reported from the *Lancet* laboratory,¹ where it was stated that the bacillus of Massol was present "in its highest state of activity." In their examinations they had found great variations in the composition of massolettes, some of them containing only streptococcus in pure culture; and the statement that "a single massolette placed in half pint of milk kept at 100° F. soon developed lactic acid" (vide *Lancet*) meant nothing, as the number of bacteria inducing the lactic fermentation was known to be very large. He was quite convinced that the majority of so-called lactic-acid preparations on the market were valueless and should be disregarded by the medical profession. In order to obtain real data as to the therapeutic value of *Bacillus bulgaricus*, it was necessary to study hospital cases in which the conditions of purity of culture could be rigidly observed. One such case had been treated in this way at the London Hospital under Dr. Wall. The case was one of colitis with very offensive stools. Benefit was believed to have taken place, but no bacteriological evidence of improvement was visible in the stools.

Dr. EYRE agreed as to the unreliability of the commercial preparations of the bacillus. Many of them had been examined at Guy's, but none were pure cultures. Sour milk was a food and drug combined. It was important to carefully select the cases which were to be treated with the sour milk. At present there was a sour-milk fashion, and it was supposed to be good for every symptom and disease. Sometimes good results were obtained in spite of the haphazard prescribing. In the remarks he was going to make he referred only to pure cultivations of the Bulgarian bacillus, and the effects which could be hoped for from the use of them. It had been shown in Metchnikoff's laboratory that the Bulgarian bacillus would produce a marked effect on the by-products of the organisms with which it was grown in symbiosis, and it had been shown by human experiment that the bacillus could be acclimatized in the intestinal canal. This being so, the Bulgarian bacillus could be used for cases in which one wanted to modify or alter the by-products of digestion which were being elaborated in the intestine. The bacillus also exerted in the intestines a powerful inhibitory action upon other organisms, notably the putrefactive ones, and also upon some of the more delicate parasites. So it could be used to restrain certain of the activities of the *Bacillus coli* and to destroy some of the bacteria present in cases of colitis. There were two kinds of colitis in which

¹ *Lancet*, 1909, ii, p. 1678.

he had seen very good results follow from the ingestion of milk soured with the *Bacillus bulgaricus*: firstly, the ordinary mucous colitis, where much mucus was passed and perhaps some blood; and, secondly, the form in which practically nothing but blood was passed, with a few flakes of mucus. The first type of colitis appeared, from the investigations of Hale White and himself, to be due to atypical forms of the colon bacillus, and seemed very amenable to treatment by the Bulgarian bacillus. In the second type of case, which was nearly always due either to the streptococcus or to the pneumococcus, one found that the ingestion of milk soured by the Bulgarian bacillus caused the pathogenic cocci to die out very quickly, even when the clinical condition had persisted for a long time. But something seemed to be required in addition to the milk cultures; the defences of the body were defective, and the defences needed stimulation by vaccines and other preparations. Some of his cases had been rather striking. In one of them the cultivations which were made from the stools gave pure growths of streptococci. Four or five days after commencing treatment with sour milk the streptococci were fewer in number, and at the end of about a fortnight they were no longer to be found, while the Bulgarian bacillus was present in enormous numbers. In another case, where the pneumococcus was the infecting organism, the patient was passing practically pure blood, and had been doing so for a considerable time. But within ten days of the commencement of the treatment the pneumococci could no longer be detected in the cultivations made from the stools. In both those cases, as also in the others, vaccines were used at the same time. But the alteration in the intestinal flora had been too striking to be attributable to the vaccine alone. It was important to give not only pure cultures of the bacillus, but also fluid cultures. The principle of administering tabloids was not to be recommended, for they introduced into the alimentary tract bacilli in a dormant state, which took time to regain their normal activity; only a few bacilli were put into the patient by that means at each dose, whereas by the fluid preparations millions of bacilli, in a vigorous and active condition, might be administered with almost every mouthful of the soured milk, and rapid acclimatization secured.

DR. ROBERT HUTCHISON said the results mentioned had been so unanimous that it was unnecessary to say anything further from the pathological standpoint. But from the clinical aspect he would say a word as to the use of sour milk and lactic acid. He was profoundly sceptical about the whole of the sour-milk treatment. It rested on a very uncertain basis; for he believed most clinicians would agree that the theory of intestinal auto-intoxication had been very much overdone, and that there was no proof which was worth anything at all that the absorption of poisons from the intestines produced many of the symptoms which had been attributed to them. In most such cases the patient was neurasthenic, and merely happened to have

gastro-intestinal manifestations as well. All careful work tended to show that, in chronic constipation, where, above all, one would expect intestinal auto-intoxication, the number of bacteria in the stools was less than normal. When he put aside that a priori distrust, and asked himself whether it gave any good results, he found his scepticism confirmed. In the last six months he had seen dozens of patients who had been taking sour milk, either on their own initiative or by the advice of their friends or that of a doctor, for various affections, and in hardly any had real benefit resulted. He admitted that some of the preparations used might not contain the Bulgarian bacilli at all; but his main point was that there was no proof that any good resulted, apart from the fact that the patients were taking a very digestible form of milk. If the ordinary patient were told to drink milk, he probably would not do so; but if told to drink sour milk, he would. If an "intestinal antiseptic" were required at all, he would rather trust to small doses of calomel, which he believed to be more effective and less inconvenient. Nothing had been said as to how long the supposed cures by lactic acid lasted; how long the intestinal flora existed without having to be replaced by fresh inoculations of the bacillus. He would like to refer also to a paper by Dr. Rosenberg,¹ who had tested the matter fairly fully in a sanatorium, where he had the patients under careful and continuous observation. He used the remedy in a large number of cases of intestinal disturbance for two years, and his conclusion was such as he (Dr. Hutchison) had stated, that there was no proof of the bacilli having done good, apart from the improved nutrition which the milk gave. If the present discussion did something to squash the sour-milk craze, which was rather discreditable to the profession, considering the way it was taken up, it would have served a useful purpose.

Dr. MILLER said that for the last year he had been treating, at the London Hospital, many cases of rheumatoid arthritis, the majority of them hospital patients, though some were private patients sent up to the hospital by doctors. Almost all of the latter had been treated with soured milk or lactobacillin, in pellet or powder form. The results of the treatment he could only vouch for from what the patients told him. The cases fell under two headings: those who had derived benefit, and those who had not. The first class had shown improvement, not as regards the rheumatoid arthritis, but as regards their general well-being, because they had become better nourished. Those who had taken the various preparations, powders, &c., had not received any benefit at all, except in what had been called the psychological respect. The few days' supposed benefit could be brought about by any new remedy. His experience, therefore, was that as a food it was sound, but that in any other respect it failed.

¹ *Archiv. f. Verdauungskrankh.*, Berl., 1909, xv, p. 458.

Dr. HERTZ said he was in almost entire agreement with what Dr. Hutchison had said. There was very little evidence that the majority of conditions which some people said were due to intestinal auto-intoxication were due to it at all; more frequently the constipation was secondary to the general condition, such as neurasthenia. He would like to hear from Dr. Herschell whether he had any chemical or bacteriological evidence that the cases of neurasthenia, anæmia, neuritis, and arthritis which he had treated with lactic-acid bacilli were due to intestinal auto-intoxication. In order to be sure that improvement in any case was due to sour milk, a bacteriological control was essential. He thought that cases of colitis which were definitely infective in origin could be benefited by sour milk. He had had a very striking case of this sort, in which the stools were bacteriologically examined for him by Dr. Eyre. The patient had suddenly become ill with a rigor, and for several months afterwards he had abdominal pain, associated with constipation and the passage of mucus in his stools. Some time after the commencement of the symptoms he had his appendix removed, but with no benefit. Dr. Eyre found that the proportion of streptococci to *Bacilli coli* was ten times greater than the normal, and that they were of a pathogenic variety. He was put on sour milk, and, as the opsonic index was found to be just over 1, it was not thought necessary to give a vaccine; he had simply the sour milk prepared with lactic-acid organisms supplied by Dr. Eyre, and daily irrigation of the colon with water. At the end of a fortnight the stools were examined again, and they were found to be practically normal. He felt perfectly well for the first time for six months.

Dr. GORDON LANE said that, so far, no one had indicated where a reliable preparation of the lactic-acid bacillus for treatment could be obtained.

Dr. LANGDON BROWN said he agreed that many of the preparations on the market were inactive; at any rate, many of those tested at St. Bartholomew's Hospital were. The only case in which he had seen a definite result traceable to the treatment, because no other method was employed at the time, was that of a man who was suffering from much intestinal flatulence; there was also much indicanuria. After the lactic-acid treatment had been carried out for three weeks he got rid of his symptoms, and the indican disappeared from the urine.

Dr. HERSCHELL, in reply, said he was glad most of the speakers were agreed as to his contentions. When he advocated the use of tabloids, he distinctly said that they must be tabloids which contained the living bacilli. He thought there was one tablet which fulfilled that condition, although Dr. Bulloch disagreed—namely, Martindale's "Trilactine." He had cultivated the bacillus himself from it, and he thought any one who did so in the proper manner would have a like result. The first of the tubes shown, that coloured

pink, was obtained from that tablet, and from that identical tube an agar plate was made with quite characteristic colonies. None of the other preparations he had tried contained the living Bulgarian bacillus, except, some months ago, the first tablets made by Bouchart, of Paris. With regard to the liquid preparations, Oppenheimer's lactigen contained the living bacillus in the samples he had examined; it had only to be cultivated on milk and it could be demonstrated with the microscope. For administration to patients he made use of a liquid culture which he prepared himself, by taking a solution containing 2 per cent. of peptone, 2 per cent. of lactose, and 1 per cent. of salt, and he inoculated that with a tube of Oppenheimer's lactigen and put it in an incubator for twelve hours. The patient took one wineglassful three times a day, and he had obtained good results in certain cases of colitis, but only where the Gram-positive organisms were in excess in the stools. And he thought the want of success in the treatment of the cases which had been recorded by Dr. Hutchison and others was due to the fact that they had not made a proper bacteriological examination of the stools, and that they had given it at haphazard in unsuitable cases. Certainly the German school disbelieved in auto-intoxication from the intestine and its association with neurasthenia; but since those views had been promulgated there had been much evidence, chiefly by French observers, that it certainly existed, and that it was marked by indicanuria, by alterations in the co-efficients of Combe and Bauman-Morac, by increase of the capillary constant, and by other things which could be observed if sufficient trouble were taken.

A Fatal Case of Poisoning by Phosphorus, taken as an Abortifacient, with Unusual Subcutaneous Hæmorrhages.

By REGINALD G. HANN and R. A. VEALE, M.B.

THE use of phosphorus by abortionists in England is now very rare; it was never so popular here as it was a generation ago in Germany, where it was by far the most common means employed either in the form of heads of lucifer matches or vermin-killer. Latterly lead has become largely used in this part of Yorkshire, and is now, practically speaking, the only metal resorted to for the purpose. During the last four years an average of about ten cases per annum of suicide by phosphorus appear in the Registrar-General's returns, though it is likely that this number should be increased by the addition of some classified as deaths from vermin-killers. In the case now recorded we infer, as so small a quantity of the paste was taken, that the girl's intention was to bring about a miscarriage, for it is notorious that suicides tend to be lavish with whatever means they select.

An unmarried girl, aged 19, on her mistress becoming aware that she was about two months' pregnant, was summarily dismissed from her situation and went into lodgings. Towards noon on the following day she took a quantity, afterwards estimated at a drachm, of a rat-poison containing about 4 per cent. of phosphorus, and within a short time complained of abdominal pain and a feeling of distension. On the next day she was unable to get up until the afternoon, and, while dressing, she vomited some clear fluid. The sickness recurred, and was accompanied by great thirst. The bowels were not moved. She took no food whatever on this day. On the third day, after drinking tea, she rose at 10 a.m., and during the morning vomited repeatedly. In the afternoon, finding her left foot swollen—evidently owing to the first subcutaneous hæmorrhage—she walked to the hospital, but, owing to some irregularity on her part, was not attended, and returned to her lodgings and had tea. She went to bed early and had a supper of bread and milk. There was thus some abatement of the symptoms on this day, as was shown by the fact that she was able to get out and also to take food. At 2 a.m. on the next day she felt so ill that she aroused the people of the house. Vomiting was frequent, blood being present in

considerable quantity; abdominal pain was severe, and a profuse uterine hæmorrhage appeared. Thirst was continuous and unrelieved by the large quantities of water she drank.

She first came under observation at 11 a.m., on the fourth day of her illness. She was semi-conscious, extremely restless, perpetually turning from side to side in bed, unable to answer questions, the only reply to attempts to arouse her being an appeal for water. Hæmatemesis was frequent and the uterine hæmorrhage copious. On examination a remarkable condition was disclosed. Below the waist-line more than half of the total skin area was the seat of enormous subcutaneous hæmorrhages. The front and back of each thigh, both buttocks, the greater part of the left leg and the right foot presented continuous patches, dark purple in colour with sharply defined and often crescentic margins. The intervening skin, as elsewhere throughout the body, was normal. There was no jaundice. No satisfactory examination of the abdomen could be made owing to the pain and the girl's restless condition. There was incontinence of urine. The bowels were not moved. The temperature was subnormal; respirations 48; pulse 120, very feeble. She was admitted into hospital under Dr. E. F. Trevelyan, but died at 7.40 p.m.—less than three and a half days after taking the poison.

The post-mortem examination was made nineteen hours later. The body was that of a heavily-built, stout girl, abdominal fat at least $1\frac{1}{2}$ in. in thickness. No secretion could be squeezed from the mammae. The large, purpuric areas were already showing signs of putrefaction, the edges becoming green, and on the right thigh there were a few bullæ filled with blood-stained fluid. It was noted that there was considerable staining around the puncture made by a hypodermic needle. The brain was normal. On opening the thorax it was found that the pleural cavities contained a small quantity of dark-coloured fluid. Much hæmorrhage had occurred into the mediastinal tissues. Numerous subpleural ecchymoses were seen. The heart and lungs were normal. There was slight excess of peritoneal fluid, hæmorrhagic in character. Most extensive retroperitoneal hæmorrhages were present, especially about the neighbourhood of the left kidney, while there were also hæmorrhages in the omentum and the mesentery. The liver was enlarged, weighing 54 oz., and was of a brilliant canary-yellow colour. The liver substance bulged from its capsule on section and presented the appearance of acute fatty degeneration. There were hæmorrhages

into the wall of the gall-bladder, which was thickened and gelatinous-looking. The kidneys were normal in size, and on section pale yellow in colour. The stomach was filled with dark fluid material; its mucous membrane appeared normal. The uterus was enlarged and contained a hæmorrhagic mole consistent with the history of a two months' pregnancy. The right ovary showed a corpus luteum. The rest of the abdominal viscera were normal in appearance. Unfortunately, no specimen of urine was obtained.

Sir Thomas Oliver, who has examined microscopic specimens from the case, writes: "The liver shows signs of very advanced fatty degeneration, a large number of the cells having been completely transformed. The kidney shows very slight changes, indicating therefore that the brunt of the poison had been borne by the liver. The fatty degeneration so marked in character is interesting seeing the short time the patient lived after taking the poison."

REMARKS.

Jaundice, due to blocking of the bile-capillaries, is present in 80 per cent. of these cases by the third day. Though its early appearance is of serious import, speaking generally it bears no constant relationship to the gravity of the disease or to the extent of the changes in the liver. It may well be that in some rapidly fatal cases, such as ours, death comes too early for jaundice to develop.

As is usual, the primary symptoms—those of gastro-intestinal irritation—were marked off from the secondary signs of toxic poisoning due to hepatic failure by a period of comparative comfort on the beginning of the third day. The only symptom which did not share in this improvement was the thirst which, throughout the illness, was persistent and distressing. We think it likely that the massive sub-peritoneal hæmorrhages stood in direct causal relationship to the abdominal pain of the latter part of the illness, and would compare this with similar conditions as they occur in Henoch's purpura and in acute hæmorrhagic pancreatitis.

The subcutaneous bleedings in their wide extent were unlike anything seen in ordinary cases of purpura, and, so far as we know, have never before been recorded in phosphorus poisoning.

Report on a Drug known as "Kangalugi."

By H. WIPPELL GADD.

DR. J. E. S. OLD, of the British Central Africa Protectorate, reported some time since to the Therapeutical Society on a native remedy for African tick or spirillary fever of man. He stated that the remedy was compounded as follows: "As many ticks (species 'Ngufi') as can be obtained are mixed with the bark of the root of a shrub called Kangalugi, and the whole roasted on a shovel; the burnt material is thus reduced to powder. When it is desired to produce immunity against the frequently fatal bites of this tick, local scarifications of the skin are made in different parts of the body, and some of the powder is rubbed in. Should a non-immune native find that he has been bitten by these ticks, he scarifies the sites of the bites and rubs in the powder in the same way, thus escaping the fever it disseminates. The root of the shrub is also used, with others, in the treatment of rheumatism, elephantiasis, the vomiting that accompanies a distended abdomen (? disease), and as an aphrodisiac (in men only). It is taken internally in the form of a decoction. When stirred up with water, a soapsud appearance is produced."

Dr. Old sent some leaves of the shrub, remarking that there are said to be no flowers or fruit, and some twigs with leaves and pieces of root. These were sent to me by the Secretary of the Therapeutical Society for examination. In order to identify the shrub botanically, I sent a portion to Mr. E. M. Holmes, the Curator of the Museum of the Pharmaceutical Society. He reported to me as follows: "At last I have identified the Kangalugi root. It is *Deinbollea Nyikensis*, Baker; natural order, Sapindaceæ; described in the *Kew Bulletin*, 1897, p. 249. I identified it by the following method: Leaves, pinnate; no round oil glands, therefore not Rutaceæ; no stipules, therefore not Leguminosæ; no resin-ducts in veins, therefore not Anacardiaceæ. I then shook up a fragment of bark with water; ample froth resulted. Not Polygalaceæ, which has simple leaves, probably Sapindaceæ. I examined the Central African genera of Sapindaceæ in Kew Herbarium. Genus *Deinbollea* was the nearest to your specimen in reticulated character of leaf, shape, and margin. Your plant was practically matched by *Deinbollea Nyikensis* in the number of the lateral veins and character of the hairs, &c. The root certainly contains a quantity of saponin."

The root was then powdered and treated in my laboratory, there being ultimately isolated from it a white powder of a crystalline character, which yields, on hydrolysis, a large amount of glucose. This is probably the saponin which the observations of Dr. Old and Mr. Holmes would lead one to expect to be found in the root. I sent a sample of this white powder to Dr. W. E. Dixon, and he reported on it as follows: "It is extremely irritant, and set up vomiting and diarrhoea from direct irritant action on the alimentary canal. In small doses it produces little effect; it is true that it has a digitalis-like action on the heart, but I have failed to show that it is absorbed in any but the smallest amounts when taken by the mouth. On the whole, it was very disappointing and quite unlikely to be of any practical value."

Kangalugi root would therefore not seem to be worthy of any prolonged research, as it is not likely to offer any advantages over other drugs already in use in European practice. It is evidently very potent, and should therefore be used, if at all, with the greatest caution.

Therapeutical and Pharmacological Section.

February 1, 1910.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

The Immunity Reaction to Cancer.

By E. F. BASHFORD, M.D.

It would imply an unusual degree of temerity were I, unsolicited, to address, on my present subject, an audience mainly interested in therapeutics. Since, however, you have been so good as to invite me to do so, I presume you appreciate that I do not come before you to talk about the treatment of cancer, except in so far as my remarks are an account of the present position of the attempt to define the direction along which a rational treatment may ultimately be found.

One of the fundamental results of the successful transference of cancer from one animal to another is the attainment of entire agreement as to what it implies. The experimental transference of cancer is effected only by the transplantation and the continued growth of living cancer-cells. Now, although it may be going too far to assert that the disease is never transferred naturally in this way, it is nevertheless certain that the transference of living cells from one individual to another is not the cause of the great frequency of cancer. I do not propose to discuss the ætiology of the disease; nevertheless, in the reactions to which I shall draw your attention you will be presented with some of the evidence proving not only that cancer in one species has no immediate relation to cancer in another species, but also that in different individuals of the same species it arises independently of

pre-existing cases. The general conception of the ætiology of the disease underlying my remarks is, cancer arises *de novo* in each individual attacked.

I shall not detail the opposition which has had to be overcome from 1902 onwards in substantiating the claim that cancer in man and animals is a similar biological process, nor shall I worry you with proofs that all the characteristic lesions of the disease can be reproduced experimentally. The first duty of those engaging in the experimental study of any disease is the reproduction of its characteristic lesions in previously healthy animals. Only thereafter may attempts to demonstrate the prevention or cure of the lesions claim justification, if the disease, as it occurs naturally, remain obdurate to attempts to modify or to end its progress.

Cancer is ubiquitous throughout the vertebrate kingdom, and before alluding to the significance of the reactions revealed by the study of suitability and resistance to the transplantation of cancer, I would direct your attention to the results of the investigation of zoological relationship by methods other than those of the systematic zoologist. The rational experimental investigation of cancer has followed upon the recognition of the importance of the biological differences revealed by the study of the hæmolysin, precipitin, and kindred reactions. By these experimental methods a scale of protein relationships has been ascertained to obtain in the animal kingdom. These reactions are elicited not only by the living cells; they are also functions of the protein substances. By means of these delicate reactions animals as nearly related zoologically as the mouse and the rat are shown to differ widely in the nature of their respective proteins.

Great expectations of a rational organo-pathology and organo-therapy were awakened when these lines of investigation were being explored for the first time, and cytotoxins were produced for special tissues on which they acted in the test-tube. When it was discovered also that the actions could be prevented by the intervention of antibodies, optimistic views prevailed as to the possibilities of treating several diseases of obscure ætiology. Cancer was naturally one of the diseases which attracted attention early, because of the hope of obtaining a serum acting upon the cancer-cells. But the wide expectations aroused at the outset of this line of inquiry have not been fulfilled in its later developments, either where they were entertained in regard to the experimental production of disease—e.g., of the nervous system—or where they were entertained in regard to the therapeutics of disease—

e.g., in the treatment of cancer. The triumphs of this line of inquiry have lain in the realms of diagnosis and of legal medicine.

In considering the stagnation which has overtaken some aspects of these lines of investigation, it is necessary to note two limitations which ought to have weighed with those responsible for pointing out a vaster vista of progress in cellular pathology and therapeutics than has actually been visualized: (1) These reactions were obtained by the introduction of tissues of one species into a strange species, and (2) they were demonstrated *in vitro*. It is certainly a startling fact that animals, not only nearly related, but also actually living on the same food, even on the pasture of the same field, build up living protein recognizable zoologically and to the unaided eye—e.g., as horse, cow, goat, sheep, hare, rabbit, and guinea-pig—and as dead protein distinguishable in the test-tube, in consequence of the specific serum reaction the protein substance of any one species elicits when introduced into the body of any other—e.g., when the blood of the hare is introduced into the rabbit, or vice versa. It is quite easy to beg the questions raised by the fundamental biological fact of proteins so different being built up from the same raw food-stuffs, by rejoining: "Of course, the starting point has not been the food, but the already living protein molecule—Jacques Loeb's autocatalysator—provided by the pre-existent cows or rabbits, &c." Whether the food is broken down into the same elements in the digestive canal of all the above-mentioned herbivora or not, the proteins resulting from the raw food-stuffs are so very different as those of the cow and rabbit, or so similar and still different as those of the mouse and rat. The preceding considerations mark obviously a big advance in knowledge gained since the days when surgeons attempted to graft the skin of rabbits or kittens on to human beings, or transfused the blood of the sheep into human patients. As regards cancer, they show how irrational was the attempt to transfer it from one species to another, except on the assumption of the presence of a cancer parasite of the existence of which experiment has yielded no positive evidence.

In general, the more remote the relationship of the species whose tissues or fluids are employed to elicit these *in vitro* reactions, the more easily are they demonstrated. The animal immunized against strange blood does not produce a hæmolysin acting on the red cells of its own species, and when a hæmolytic immune-serum is not strictly specific—i.e., acts on the blood of more than one species—its action can be simplified, as regards any particular species, by employing that species to yield the hæmolytic immune-serum. Whenever it has been attempted

to elicit a corresponding reaction *in vitro* after introducing the blood of one individual into another of the same species, dubious or completely negative results have followed, with the single exception of a series of experiments performed on goats; the feebleness of the reaction (isolysin) demonstrated as compared with that obtainable with strange species (heterolysin) made, at the time when these experiments were performed, a deep impression upon me which lasts to this day. The attempt to produce experimentally a corresponding reaction *in vitro* by introducing again into an individual some of its own blood (autolysin) has failed altogether, although it has been claimed that the corresponding reaction has been obtained for spermatozoa (auto-spermotoxin).

A corollary to this line of investigation arose in the difficulty of obtaining *in vivo* the cellular reactions apparently so specific *in vitro*. Sera highly cytotoxic for a specific tissue *in vitro* were without action, or, at any rate, any action exhibited was not specific when tested *in vivo*—e.g., auto-spermotoxin and hetero-spermotoxin did not attack the spermatozoa *in vivo*, but only *in vitro*. An exception to this generalization appears to be offered by the action of gastrototoxic serum; but even in this connexion I should be inclined to sound a note of warning. Gastric ulcers appear to have followed only upon the injection of gastrototoxic serum, either intraperitoneally or locally. Although, in regard to the success attending its employment for this specific purpose, gastrototoxic serum appears to stand alone among the heterologous cytotoxic sera, I am unaware of any evidence of the production of a specific gastrototoxin either by homologous or autologous inoculation of gastric mucous membrane. With regard to cancer, the sera obtained from animals of one species after immunization with the tissues of malignant new growths of another species have no reaction *in vitro* distinguishable from the well-known hæmolysin and precipitin reactions. The reactions of an "anti-cancer" serum obtained by immunizing one species with the cancerous tissue of another species are in all respects parallel to those by which blood relationship has been established, and are equally subject to limitations of their action *in vivo*. It may be stated with confidence that all claims to influence the growth of cancer by any such "anti-cancer serum" are based up to date upon fallacies of one kind or another. One thing stands out of all others—viz., the difficulty of demonstrating *in vitro* any reaction with serum obtained after the introduction of homologous or autologous tissue. The search for hæmolysins, cytotoxins, and corresponding reactions¹ demonstrable *in vitro* due to the

¹ The isoagglutinins are probably an exception.

production of anti-bodies, becomes uncertain when reactions are sought between individuals of the same species, and it becomes altogether hopeless when it is sought to induce reactions between an organism and its own tissues.

It is at this point that the reactions revealed *in vivo* by the experimental study of cancer take up the thread of the progress of experimental biology. In contrast to the hæmolytic and kindred reactions obtained by heterologous immunization, the reactions having direct bearing upon cancer are only manifested *in vivo*, and they are elicited not only by immunization with homologous but also with autologous tissue. As I have said, we have good reasons for assuming the autochthonal and *de novo* origin of cancer; wherever it occurs the cancerous tissue may be regarded as part and parcel of the individual attacked. I ask you to consider the reactions of suitability and resistance to the implantation of cancerous tissue into other animals, from the standpoint that the study of the lesions of experimental cancer, and of the means of modifying them, is a necessary preliminary to unravelling the phenomena of spontaneous cancer. As I have often emphasized, the conditions differ in the two cases, because in experimental cancer we observe the behaviour of the tumour-tissue of one animal in a multitude of other animals, whereas in spontaneous cancer the tumour is tissue of the animal attacked.

In the first place I would point out the extreme delicacy and the demonstrative clearness of the reactions revealed by using living cells and living animals as indicators of biological differences, either naturally existing or experimentally induced. Whereas the specific nature of the precipitin reaction revealed unsuspected differences between the body fluids and the tissues of even nearly related species living on the same food, the transplantation of cancer has revealed differences in animals of the same species racially distinct. Further, it has revealed differences in animals of the same race but of different age, and also differences in animals of the same race and age. It has defined still more subtle differences by revealing those obtaining even in animals of the same race and age when naturally afflicted with cancer of the same organ—e.g., the mamma; it has demonstrated that different tumours of a single organ elicit a specific connective tissue and vascular scaffolding, thereby revealing differences between cells histologically indistinguishable. In addition, the transplantation of cancer has made the study of the *vita propria* of the cancer-cell possible. It has revealed, among other facts, the multiplicity of varieties of cancer-cells which may

arise from the normal epithelium of a single organ. I must pass over this aspect of the experimental study of cancer, notwithstanding that it possibly contains the germ of a full comprehension of the nature of the disease. I will allude only to a yearning expressed thirty-five years ago: "What a subject for Darwin would be the cells of a cancer if only they were tangible; how the immortal pigeon would be completely eclipsed, while the hungry pathologist would be filled with food, if only we could observe the variation of tumours under judicious cultivation!"¹ The goal yearned for in this dream of thirty-five years ago, to which Sir Samuel Wilks, being aware of the trend of our work, has drawn my attention, is no longer an unattained ideal, but an accomplished fact to-day. Some seventy distinct varieties of carcinoma descended from normal mammary epithelium, as well as their sub-varieties, are under observation in the Laboratory of the Imperial Cancer Research Fund. Ignoring the wider significance of the experimental study of its *vita propria*, I will summarize the practical importance of the cancer-cell having been made "tangible" by asserting that no method at present available reveals, both for organisms and for living cells, such fine biological differences as those which are so clearly demonstrated by implanting the living cancer-cell into animals, where it can be subjected to many experimentally induced conditions.

The fact is of great biological moment that cancer of one species will not grow continuously in another species; but from the standpoint of immunity, it is to be regarded simply as a consequence of the cancerous tissue being merely the protein of a strange species. Therefore, it is not surprising that animals after preliminary treatment, either with the cancerous or normal tissue of a strange species, are not thereby made resistant to a subsequent inoculation of cancer of their own species. This phenomenon is demonstrated in the accompanying figures,² where rats previously "treated" with sarcoma of the cat and mouse respectively are shown to remain suitable for the inoculation and growth of rat sarcoma or carcinoma. Similar negative results, as regards immunity, obtain if animals are first treated with normal tissues of strange species. The accompanying figures show how rat skin employed in this way is impotent to protect mice against the implantation of squamous-celled

¹ James F. Goodhart, M.D.: "On Cancer as illustrated in Ichthyosis of the Tongue and Allied Diseases resulting from Prolonged Local Irritation," *Guy's Hosp. Reports*, 3rd Ser., 1875, xx.

² The paper was illustrated by a number of lantern slides, but corresponding figures do not accompany its publication in the *Proceedings* of the Section.

carcinoma of the mouse. Other figures illustrate the nature of the change ensuing after a preliminary inoculation of normal or of tumour tissue of a strange species, as it is revealed by examination of the site where subsequent re-inoculation is made. The reaction elicited by the preliminary inoculation is reinforced at the re-inoculation, and, in addition, influences directly lethal to the introduced tissue are obviously in play. This is only a consequence one would expect from a procedure which is parallel with that employed to obtain heterologous immune-sera having a cytotoxic action *in vitro*; but it is far from being an immunity reaction to cancer *qua* cancer.

Cancer tissue will only grow continuously in other animals of the same species as that in which the tumour developed, where alone the necessary food-stuffs are supplied. The question of pabulum is, however, not the only matter of moment, as the following considerations show. When a malignant new growth is removed from the individual mouse in which it developed, and implanted into a number of healthy mice, rarely do the implantations take in more than 12 per cent., and the results may be entirely negative in as many as 900 attempts. It has been customary to explain the negative results of this homologous transplantation by assuming that the majority of healthy animals are naturally resistant to the inoculation of cancer of their own species. But as an interpretation of the facts the term "natural resistance" is nothing more than a meaningless phrase. In addition to the two variables which were at once recognized—viz., the varying qualities of the animals and the varying qualities of the tumour cells—quite a number of others have already been defined. Such investigations, however, bear more on the nature of cancer than on the induction of immunity, and I pass them over after merely intimating that, although it is difficult, and sometimes impossible, to obtain the continued growth of a tumour in normal animals, recent extensive re-investigations of the results of transplanting tumours back again into the animals in which they developed have greatly improved upon the results previously recorded. They have demonstrated that auto-transplantations are successful, under given conditions, in 95 per cent., and thereby the frequency of large metastases in animals spontaneously affected is harmonized with the difficulties in obtaining growth by implanting the tumour of an animal naturally affected into normal animals, or into other animals naturally affected with cancer. The next series of considerations have some bearing upon why transplantation into other animals of the same species (homologous transplantation) so frequently fails.

Reactions inimical to the implantation and the continued growth of cancer-cells are induced in living animals by preliminary treatment with homologous tissue, either cancerous or normal. The figures illustrate how high a degree of resistance may result to subsequent inoculation after tissue has been absorbed in this way. More interesting still is the fact that this form of resistance may be induced concomitantly with the establishment of a tumour, and indeed may be so effectively induced as to cause, on the one hand, a large proportion, or even all the tumours developing, to exhibit only transitory and not progressive growth, or, on the other hand, to render the animals resistant to a secondary inoculation, when the tumours from the first inoculation continue to grow. This resistance presents many interesting features. In the first place it is specific; the resistance induced by the absorption of tumour tissue is most effective against a re-inoculation of the tumour absorbed, and may be almost ineffective against other tumours. Thus, a complete resistance to the implantation of a carcinoma is compatible with a suitability for the implantation of sarcoma. The preliminary absorption of different normal tissues is of varied efficacy. One may say skin protects best against the inoculation of skin cancer; but skin also protects best against the inoculation of mammary carcinoma, and the apparent relation between the protection induced by normal tissue as against tumours of the corresponding histological structure cannot be referred only to similarity of histogenesis without important reservations. The perfect protection mouse skin induces against squamous-celled carcinoma of the mouse, and the absence of this action in the case of skin of the rat, is very striking; but the efficacy of mouse skin as a protector depends—apart from the main factor that it is mouse tissue—also on such technical factors as those of dosage, which obscure, if they do not outweigh, the importance of the histological relationship of the skin of the mouse to squamous-celled carcinoma of the mouse. The spleen also induces a high degree of protection against epithelial tumours. The analysis of the induction of artificial resistance can be carried still further. The subcutaneous inoculation of tissue removed from an animal and immediately re-inoculated into itself effectively induces protection, as illustrated, for the autologous inoculation of spleen.

It has thus been shown that there is a parallel in the behaviour of cancerous tissue and of normal tissue of the same species, in regard to their powers of inducing protection against inoculation; and, further, it has been shown that it is immaterial whether the normal tissue is provided by another animal (homologous inoculation) or by the very

animal itself (autologous inoculation). The ease with which these reactions are obtained *in vivo* and their demonstrative clearness are in marked contrast with the difficulty of obtaining "isolysin" reactions *in vitro* and the failure to obtain any corresponding "autolysin" reaction at all, either *in vitro* or *in vivo*. The mechanism of resistance after immunizing with homologous normal or tumour tissue is also in marked contrast to that described above as obtaining in the case of the heterologous reactions *in vivo* after preliminary treatment with cancer or normal tissue of strange species. It has been impossible to obtain any effect *in vitro* of the serum of animals immunized with homologous tissue or tumour which is not possessed in equal degree by normal serum. All attempts to detect anti-bodies transferable from one individual to another, or from the pregnant or suckling mother to her offspring, have failed. The quantitative relations obtaining between the degree of immunity and the dose of material which induced it, as well as between the degree of immunity and the doses of tumour against which it is effective, speak for the intervention of active substances or forces of some kind which can be used up. There is also some evidence of direct interactions taking place in the body fluids of immunized animals so that the degree of immunity can be lowered. Although it is difficult to conceive that alterations in the body fluids are not in part responsible for the change, there is no evidence of a direct lethal action on the cancer-cells, which remain alive the longer the nearer they are to the tissues and fluids of the immune animal, exactly the opposite being the case for heterologous inoculations, as described above. The change manifests itself, during the process of immunization, by a general reaction (plasma-cell reaction) of the entire connective tissue of the animal. When immunity is established, it manifests itself in a paralysis of the chemiotactic powers of the cancer-cells; when implanted into an immune animal, they fail to elicit the connective tissue and vascular scaffolding necessary for their growth into a tumour. The result is the contrast between the appearances at the site of inoculation in a normal and in an immune animal, as depicted in the accompanying figures.

For the further elucidation of the process extensive studies have been necessary on the consequences following the inoculation of the protein substances both of normal and of cancer-cells, as distinct from the inoculation of the living cells themselves. In this respect, also, there is an entire parallel between the behaviour of the proteins obtained from normal and cancerous tissue. They do not induce immunity, and thereby yet another distinction is established between the hæmolytic,

precipitin and kindred reactions, elicited by heterologous inoculation and demonstrable *in vitro*; because the latter are independent of the vital integrity of the tissue inoculated. In this connexion it may be permitted to draw attention also to the difference between the immunity reactions to many bacteria and their products. Leaving out of consideration those cases in which soluble toxins can be separated from the organisms, the latter reactions, with some exceptions, are not usually or even generally regarded as dependent upon the inoculation of living organisms. It might appear that the loss of power to elicit immunity to cancer by the inoculation of homologous or autologous protein, as distinct from living cells, was merely the consequence of the indifferent nature of the proteins when re-inoculated into animals of the same species, or even into the same individual. That this is not the explanation is proved by the fact that the proteins when so inoculated are not indifferent, but may actively modify the animals in the direction of rendering them more suitable for inoculation. Here I am tempted to make a digression into bacteriology and to ask the question: Does the inoculation of bacteria, *killed* by heat or otherwise, always have consequences only milder but otherwise identical with the inoculation of living bacteria? I am aware it is generally assumed that there is only a quantitative, and not a qualitative, difference in the reactions to living or dead bacteria which are the specific cause of well-defined diseases, but the marked qualitative differences in the effects following the inoculation of the living and the disintegrated cancer-cell give cause for thought. I express no opinion on this matter beyond recommending it to the consideration of others more competent than myself to deal with it. But I do hold very strong opinions on another matter—namely, on the indiscriminate practice of what, with reference to cancer, is erroneously styled vaccine-therapy. Some of these procedures practised with organisms, perhaps carelessly killed, may, in unskilful hands, have consequences exactly the reverse from those anticipated, having regard to the fact that hypersensitiveness, and not immunity to the inoculation of cancer, can be induced by heterologous tissue and other agents. It has been experimentally demonstrated that as yet no outlook is opened on the therapeutics of cancer by employing killed cancer-cells, tumour extracts, or vaccines of supposititious cancer parasites. The employment of such empirical means as vaccines of *Bacillus neoformans*, of various cocci, of antiyeast, and other heterologous antisera as curative agents for malignant new growths has no scientific justification whatsoever. These methods of treatment are merely modern forms of empiricism. The claim has

been made that the employment of the mixed toxins of streptococci and *Bacillus prodigiosus* is justified on experimental grounds. The experiments relied upon were performed on dogs affected with growths having a histological structure like that of lympho-sarcoma. These growths are, however, infective venereal tumours, and fundamentally different both in their clinical history and their pathology from the sarcomata and carcinomata, whether occurring in dogs or in man.

Throughout the sequence of observations recorded above on the parallel behaviour of normal and cancerous tissue you will have noted that the tumour tissue used, whether for preliminary treatment in order to immunize, or for testing the animals after treatment, has usually been derived from another animal. You will have noticed also that I have referred only to the effects obtained in normal animals, and not to any effects obtained in animals already bearing tumours. This is because our observations are advancing only slowly from one sure step to another. As yet they have yielded unequivocal results only under the experimental conditions above described. It is, however, obvious that if cancer arises *de novo* in the individual attacked, then the problem of immunity to cancer, as defined by the experiments already described, resolves itself into the problem of immunizing an animal against its own tumour tissue, and, further, involves the problems of modifying both the growth of tumours already existing and of immunizing animals against the dissemination of tumours when present. The interpretation of immunity to the implantation of cancer as a "vaccination" against the natural onset of the disease, involves a grave misunderstanding of the significance of the observations. I have often emphasized the necessity for awaiting the application of modern experimental methods to animals bearing spontaneous tumours, before drawing any deductions as to the bearing of the results on the cure or prevention of cancer occurring naturally. Some years ago I re-enforced this warning by pointing out that mice which are effectively protected against the implantation of cancer may develop tumours of their own spontaneously. The need for caution must be pointed out still more emphatically to-day, since as the number of investigators who have observed immunity to implanted cancer increases, so also does the number of voices proclaiming the possibility of "vaccinating" human beings against cancer increase. I would not impress you as being pessimistic, but I would remind you of the years that have usually elapsed before the methods successful in controlling or curing diseases induced experimentally in animals, have developed into methods which could be successfully applied

to man. The systematic experimental study of cancer is of scarce seven years' duration. The definite results foreshadowing others of possible therapeutical bearing have been obtained only in the case of *normal animals* made to bear transplanted tumours obtained from *other animals*. It is possible to prevent any implantation from taking in a normal animal, but a conundrum not yet satisfactorily solved is, why the means which succeed so well in normal animals are very much less certain, and, under given conditions, usually fail to prevent an implantation into an animal already bearing a transplanted tumour. These investigations have, of course, important bearings upon the continued growth of a tumour once it is present, and possibly even more direct bearings upon dissemination. Our attacks on this problem now extend over the four years which have elapsed since experiment disclosed that it existed. Persistent endeavour is being met with gradually improving results: the growth of an established tumour can be somewhat modified by the inoculation and absorption of tumour tissue, and secondary inoculations in animals already bearing tumours can be prevented by the presence of an active resistance which has been induced in a similar way. The questions here are not only those pertaining to the influence a growing tumour may exercise upon its host, but are also those of mastering important matters of technique, and of hunting for and utilizing that material which affords opportunity for arranging the suitable experimental conditions. I do not propose to go beyond stating there is ground for hopes that the difficulties will be overcome as many others have been. There is encouragement in the indications that the growth of established tumours can be experimentally retarded as well as in the evidence that secondary implantations (artificial metastases) can be prevented in tumour-bearing animals. In both respects the indications are as suggestive and encouraging as those which five or six years ago presaged the now undisputed achievement that the implantation of cancer can be prevented with absolute certainty. At present no such definite conclusion can be drawn as to the prevention of natural metastases. The control of transplanted tumours and of the elaboration of methods of immunizing a tumour-bearing animal against a secondary inoculation (artificial metastases) will probably precede the formulation of what the immunity reaction to cancer precisely involves. This being so, it will be premature to enter into any lengthy discussion of our experiments on animals naturally affected with cancer. The large number of such tumour-bearing animals at our disposal has already made many preliminary observations possible, but till the above problems

are solved we are working more or less in the dark, and it may be along entirely erroneous paths. The frequency with which the cure of transplanted tumours takes place as contrasted with the rarity of the occurrence in mice spontaneously attacked presents a problem the meaning of which is only dawning upon us. Out of some 400 mice affected with spontaneous cancer, the clinical course of which has been followed with the greatest care for weeks or months, we have observed unequivocal evidence of natural healing—circumscribed or complete—only some three or four times, whereas transplanted tumours may disappear in any proportion up to 100 per cent., according to the nature of the tumour and the technique employed. However, natural healing of spontaneous tumours does occur both of the primary growths and of the metastases. Its rarity is no evidence against an immunity reaction to cancer existing—it certainly does exist—but its final elucidation will probably be ascertained by studying the reactions occurring within the body of an animal bearing a tumour which is part and parcel of its own body. Experiment has already given us the clues I have mentioned, and I hold it is not a vain dream to anticipate the experimental discovery of others as yet unsuspected.

I have illustrated how the problems of the immunity reaction to cancer are being narrowed down; to recall only one instance, I showed you that the absorption of an animal's own tissues can modify the animal so fundamentally as to render it absolutely resistant to the implantation of cancer. Were this the only new fact ascertained it would of itself introduce fresh conceptions into pathology and biology. I need not allude to the far-reaching importance of the many entirely new facts that have been revealed by the study of homologous and autologous inoculations; they have opened up an entirely new chapter in the biological analysis of the living organism. We shall learn more of them in further attempts to define the immunity reaction to cancer, and, if years elapse before this goal is attained, I venture to believe that the experimental study of cancer will, in the interval, have made a lasting impression upon pathology and a still greater one upon general biology.

Proprietary, Patent, and Secret Remedies.

An Address Introductory to a Discussion on the Subject.

By W. E. DIXON, M.D.

THERE can be no effect without a cause, and, as I shall presently give you evidence to show that the quack-medicine traffic has been growing by leaps and bounds in this country during the last fifty years, it is well before discussing its nature that we should understand the causation of the increase. During the last half century medicine has been concerned almost entirely with the causation, diagnosis, and prevention of disease; until the last few years little accurate knowledge of the action of drugs had been obtained, and a condition not far removed from chaos seemed to reign in the minds of many physicians, some of whom openly avowed their disbelief in the efficacy of drugs in disease, for practically all therapy was empirical. And now, when the dawn of a new age is thick upon us and empiricism is being replaced by rationalism, it is hardly a matter for wonder that there are those of us who would substitute the laboratory for the bedside in all pertaining to therapeutics; others who would rely entirely on the bedside; and a third group of ardent and enthusiastic believers in all new products and methods of treatment of disease. Some regard baths, mineral waters, electricity, or radium as the panacea for all disease, and others put their trust in the lactic-acid bacillus, calcium, or whatever other drug is in vogue at the time. In such conditions at the very dawn of pharmacology can it be wondered at that the quack-medicine vendor should thrive, that the pharmacist's shop should be stocked with bile beans, Beecham's pills, and tinted soaps until it resembles a bazaar, and that the manufacturing chemist should find it a paying business to deluge the medical profession with pamphlets informing him of the best drugs to employ in disease and the best way to prescribe them? Indeed, almost all I say in this paper concerning the introduction of new proprietary drugs must be traced to the failure of the medical schools and universities in this country to promote proper departments of pharmacology.

DEFINITIONS.

Before proceeding further it may be advisable to note some of the terms in common use. *Quack* medicines was a term at one time given to nostrums sold publicly in the market-place before such time as their proprietors became millionaires. The term *Patent* medicine is a misnomer, since, as popularly used, it does not apply to products protected by letters patent under the great seal, but to proprietary remedies recommended in the treatment of disease. Anyone is at liberty to make or sell such remedies who pays for the licence, which costs five shillings. A *true patent* cannot be held as valid unless there is something novel and useful about it, and it seems now to be certain that a drug or chemical substance cannot be patented, but only the process of its manufacture. It has further been decided in the courts of law that if a patent is obtained for the preparation of a certain drug, and if a characteristic name or *trade mark* is given to that drug, the State grants fourteen years to the proprietors for the sole right of manufacture, after which time the patent lapses with the trade mark, and both become public property.

Proprietary remedies may be divided into (1) secret, for which a stamp duty is fixed, beginning at a penny-halfpenny for each shilling retail price; (2) those in which the composition is disclosed (these escape the stamp duty even when they are recommended for the treatment of certain diseases if sold by pharmacists); and (3) patent medicines proper. The owners of proprietary medicines rarely take out a patent: first, because there is nothing novel or useful in their nostrum; and, secondly, because the formula would have to be published, so they apply to it a distinctive name or trade mark. A trade mark need not apply to anything novel or useful; indeed, the substance to which it applies is frequently some common chemical bought by the proprietor ready made. A trade mark, unlike a patent, does not apply for fourteen years only, but is valid for ever. So that, if a proprietor sells a drug which contains no scheduled poison and no large amount of alcohol, and if it is stamped, he possesses absolute proprietary rights and absolute secrecy for ever.

SECRET REMEDIES.

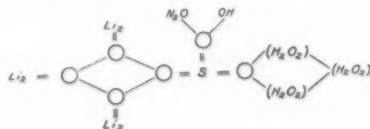
With regard to secret remedies, it is quite unnecessary for me to point out their evil effects, so I will enumerate two or three typical examples showing how they are of direct interest to the medical man. First, there is the bile-bean bubble, whose proprietor, Fulford,

accumulated a vast fortune from a credulous public. I quote this case to illustrate how the press in this country is in the grip of the patent-medicine vendor on account of huge advertising contracts. The Court of Session in Scotland in February, 1905, was concerned with the bile-bean case, in which there was a ruthless exposure of the tricks by which it was exploited on the public. The Press in England, however, was silent on the matter, so that the English public never heard of the case, and are as ignorant of bile beans to-day as they were then. If the company had happened to be a small, non-advertising firm, the evidence would not have been suppressed, as was shown in the case of *Tucker v. Wakley* and another. The perfectly harmless type of nostrum is illustrated by Munyon's kidney cure. The label says that it cures Bright's disease, all urinary troubles, and pain in the back or groin from kidney disease. According to the analysis of the British Medical Association, the pilules consist of white sugar. Tasteless quinine, another drug of this class with which, not so many years ago, physicians "cured" their malarial patients, was found to consist of powdered gypsum.

Another type of quack has learnt that much personal benefit may accrue by catering for the visionaries in our profession. The uric-acid craze, for example, is dying hard, and yet I doubt if many of us would have even heard of uric acid had it not been relatively insoluble in water; but, as it is, we have the *Uric Acid Monthly*, and its advocated specific, Thialion. According to the report of the American Medical Association, its composition is a simple mixture, as follows:—

Sodium citrate	58.6	per cent.
Sodium sulphate (anhydrous)	26.6	"
Sodium chloride	3.3	"
Lilium citrate (anhydrous)	1.8	"
Water	9.7	"

The proprietors, however, give its formula as shown graphically, a piece of absurdity which would not impose upon anyone with the slightest chemical knowledge. The easy gullibility of those medical men in this



Graphic formula for Thialion.

country who have been imposed upon by the *Uric Acid Monthly* must be regarded with considerable satisfaction by the Vass Chemical Company.

Antikamnia is familiar to us all; we have all received pictures, samples, and pamphlets. The advertising literature says:—

The well-known nerve-specialist, Dr. Harley, in an interview published in the London *Daily Express* says: "I have treated more than one American for nervousness and 'brain fag' directly due to their incessant energy. I had a young man in here this morning who complained of headache 'in the back of the neck.' He was threatened with congestion of the brain, and seemed somewhat aggrieved when I told him he had been trying to do too much."

The following is another testimonial from Dr. Caleb Lyon, an old Bellevue practitioner. In referring to antikamnia and codein tablets, he says:—

"In my practice they accompany the maid from her virgin couch to her lying-in chamber, assuaging the perplexities of maidenhood and easing the trials of maternity with most gratifying results. I earnestly hope that the proprietors of this valuable remedial agent will keep it up to its present standard of purity and excellence."

I must really apologize for quoting such stuff, but it serves to show the kind of evidence which the manufacturers consider good enough for medical men. Antikamnia, as sold in this country, according to analysis, consists of:—

Acetanilid	67.75 per cent.
Caffeine	4.88 "
Citric acid and sodium bicarbonate by difference	25.36 "

(Quoted from the *Journal of the American Medical Association*.)

The composition of the American article since the Food and Drugs Act came into effect there is different, phenacetin being substituted for acetanilid. I give this substance as another example of a secret remedy used by some practitioners in this country which contains a drug—acetanilid—which they would probably never dream of prescribing, but would always prefer to substitute the less dangerous drug phenacetin. These examples, picked out haphazard, are sufficient for my purpose to show that the medical man, like the layman, is under the thrall of the secret-medicine vendor.

CAUSE OF NOSTRUM TRAFFIC.

I propose now to deal with the cause of the success of these nostrums, and the first essential is that of secrecy. Secrecy is a device to deceive, and is quite unnecessary for the protection of any legitimate remedy.

It has been argued that the physician should be acquainted with the action of a drug and not its composition. Of course, this is simple nonsense, as it is only by the composition that the action can be known. It should also be remembered that the secret-remedy vendor sells either to the medical profession or to the general public, but rarely to both at the same time; not infrequently they begin by advertising to the medical profession only, but so soon as the general public have become acquainted with the drug the advertising to the profession ceases, and advertisements appear in the lay papers addressed directly to the general public. In England, for example, Kutnow's powder, which "dissolves and eliminates uric acid," is advertised in the lay Press, and free samples are sent to all who write for them. In the United States, however, according to the Council of the American Medical Association, Kutnows advertise to the medical profession only.

Testimonials and advertisements are of paramount importance for the sale of nostrums. A catch-word is a great asset; thus there is "purgen," a fancy name given to the well-known chemical, phenolphthalein. Analgesin is another such word given for antipyrin; diabetin, a word which has, I believe, now been abandoned, was formerly used for lævulose. Sometimes the name may be completely misleading as to the composition; thus Marienbad tablets "prepared from the prescription of Professor Ritt von Bäsch, M.D.," are freely distributed as samples among the medical profession, and no doubt the silver-coated tablets in the gilt-trimmed box labelled "Marienbad" are not without effect. They consist of aloes, rheubarb, podophyllin, cascara, and belladonna, and represent a common type of vegetable pill prescribed daily by physicians. Other means adopted by the quack-medicine vendor are clever advertisements with plausible argument and constant reiteration. Think of the skilful picture advertisements of Eno's fruit salt, a powder found by analysis to consist of:—

Sodium bicarbonate	50 per cent.
Sodium bitartrate	15 "
Free tartaric acid	35 "

It is, in fact, very similar to the common Seidlitz powder.

Letters from medical men are often published, and occasionally proprietors try to make out that the Government stamp is some guarantee of quality. Moreover, it must not be forgotten that, as these drugs are not under the Food and Drugs Act, there is no requirement that their composition should be fixed; and, as a matter of fact, the composition

is sometimes altered. Antikamnia, at the present time in America, is made with phenacetin, but that sold in England is still made with acetanilid.

NON-SECRET PROPRIETARIES.

Proprietary remedies not of a secret nature are those with which the medical profession are mainly concerned; and for the introduction of some of these medical men and the public are indebted to some of the great manufacturing houses which have fully-equipped pharmacological laboratories. Adrenalin, antipyrin, arsacetin, chloral, cocaine, veronal, eserin, the nitrites, novocaine, stovaine, strophanthus, are a few of the remedies which owe their introduction to the pharmacological laboratory. Indeed, it is not too much to say that no drug of importance has been introduced into medicine within the last twenty years except through the laboratory. What, then, can a manufacturer, who has discovered through his pharmacological laboratory a substance which promises to be useful in medicine, do in order to introduce his new drug? He may, of course, take out a patent for the process of manufacture and give the substance a distinctive name, in which case, if his patent is valid, he will have the sole right of manufacture for fourteen years, which is sometimes extended. Whether this is a sufficient reward or not I shall not attempt to decide. He may, on the other hand, refrain from taking out a patent and apply to his substance a distinctive name or trade mark, which will remain his private property always.

Not infrequently a manufacturer discovers therapeutical properties in a well-known chemical compound, and after a number of clinical experiments he persuades the medical profession to prescribe his substance under the fancy name which he has given it. Other manufacturers quickly get to know what is going on, and place the same substance on the market under yet another registered name, either devised on purpose or taken from a number of names previously registered and kept in stock for emergencies; the current methods of naming new drugs have led to the greatest confusion and frauds upon the physician and general public. Aspirin, acetylsal, xaxa, saletin, salacetin, are some of the names by which acetyl-salicylic acid is known; it is a serious matter to the retail chemist, as he may be required to stock all the different samples of the same drug to meet the views of different prescribers. Aspirin was the original substance introduced into medicine as the result of pharmacological investigations, and around which the literature gathered; the other names are parasitic, and followed later. If a right to use a

fancy name is legitimate, this should be confined to aspirin. As an example of old drugs introduced into medicine under new names we may take atoxyl, which was prepared so long ago as 1863 by Béchamp, and for which numerous fancy names have since been given. Much confusion has been brought about by the introduction of anti-febrin in 1885 for a very well-known chemical body—acetanilid. It is especially a matter for regret that many firms have made a practice of using names and trade packages which will lead to the introduction of their goods directly to the general public.

STATISTICS.

Before passing on to discuss the attitude which we should take up towards this traffic, I think a few statistics showing the extent of the evil may not be amiss. In the first place we should remember that in no country are more quack or secret remedies advertised to the general public than in this; they number, roughly, 10,000, and about the same number are advertised directly to medical men. In 1894-5 31,592 licences at five shillings each, for the sale or manufacture of proprietary nostrums, were issued. In 1904-5 this number had increased to 40,734, yielding over £210,000 to the revenue. Stamp duties were first imposed in 1793, not with the object of limiting the sale of proprietaries, but for revenue purposes, and they were supposed to yield £15,000. The increase in revenue since then is shown in the following table:—

1860	£43,366		1890	£217,267
1870	£72,353		1900	£288,827
1890	£135,366		1906	£324,112

This shows that the stamp duties yielded formerly only 2 or 3 per cent. of what they now yield, and it is obvious that they have in no way acted as a deterrent to the sale of quack nostrums.

LAWS.

In deciding the best plan of dealing with the evils which have been enumerated it may be well to examine those Acts which are directly or indirectly concerned with the subject.

The Merchandise Marks Act makes it a criminal offence to apply with intent to defraud a false trade description to anything which is subject to trade manufacture or merchandize. The Liverpool Corporation prosecuted certain retailers of proprietaries for selling goods falsely

described or obtaining money under false pretences. If only this Act could be altered so as to make it compulsory for the local authorities to take action, much good might be done; but, as it is, the Act, so far as the present purpose is concerned, remains a dead letter.

The Sale of Food and Drugs Act, 1875 and 1899, says that no person shall sell, to the prejudice of the purchaser, any article of food or any drug which is not of the nature, substance and quality demanded, but an offence shall not be deemed to be committed in those cases where the drug or food is a proprietary preparation. So that the secret-medicine vendor is deliberately placed outside this Act; his ingredients need not be pure, his tartaric acid may contain lead, and no prosecution will follow; it is the registered and qualified pharmacist who is compelled by law to sell pure drugs. A representative meeting of the British Medical Association suggested that proprietaries should be labelled with the name and quantity of each constituent, and that the label should constitute a warranty so that the vendor could be prosecuted under the Food and Drugs Act.

The Pharmacy Act of 1868 applies to proprietaries containing scheduled poisons. Proprietaries containing such poisons must be labelled poison, with the name and address of the vendor, and must be sold by a registered pharmaceutical chemist or in the shop of a registered chemist or of a limited company. The Act does not apply to medicines of Letters Patent. Winslow's Soothing Syrup is an example of a typical poisonous proprietary. Owbridge's Lung Tonic formerly contained a scheduled poison, but the composition was changed so as to free their sale from the restrictions of the Pharmacy Act.

The Spirits Act dealing with the sale of strongly alcoholic preparations, medicated wines, and the like, makes an Excise licence necessary, except for physicians, chemists, &c.

POLICY.

The methods which have been proposed to eradicate quackery in its widest sense may be divided into two kinds. Those for the secret nostrums we may consider first. There can be little doubt that if it were made compulsory for the composition to be placed on the packet of each proprietary sold, the greater evils would soon cease. Secondly, the Merchandise Marks Act might be altered, making it compulsory for local authorities to act, and last the exemption clause in the Sale of Food and Drugs Act might be deleted. These are the ideals at which we

should aim, and I place them before you to-day for your consideration and discussion. You must remember, however, that vested interests are at stake and most governments are chary of interfering with these. Also the whole of the press may be regarded as antagonistic.

In the case of the non-secret proprietaries, our position is still more delicate. If a firm or individual introduces a new drug into medicine, he should have all the protection and benefits for his discovery that we can give him, but it is hardly fair that the patent should last for ever, any more than the copyright of a book should, and perhaps the fourteen or twenty-one years which the law at present gives him as sole manufacturer of the article in question by the processes he has patented, meets the justice of the case. Many firms have introduced old and well-recognized chemical substances into medicine under a trade name; if the drug is prescribed much, other firms introduce the same substance under other names, until we get into a state of hopeless confusion. This confusion of names was one of the reasons for the publication by the Pharmaceutical Society of the Codex, in which a simple name is given for each chemical substance which was not actually patented.

Finally, it must be remembered that much of the cure for these evils lies in our hands. We should never prescribe a secret remedy; if it has any beneficial action it must be due to one or more well-known drugs which can be easily ascertained. I think we ought not to prescribe a compound medicine of a proprietary nature, especially such things as compressed tablets, in the original packages: it is a direct means of encouraging self-drugging. We ought not to prescribe a drug with a registered fancy name unless it is held by Letters Patent, but refer to its synonym and prescribe it by its chemical name, unless distinct reasons exist for prescribing the original product. It is to be hoped that some authoritative medical body, such as the British Medical Association, will see fit to publish to the profession before long such a list of synonyms.

(The discussion was adjourned until the next meeting.)

Therapeutical and Pharmacological Section.

March 1, 1910.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

Discussion on Proprietary, Patent, and Secret Remedies.¹

MR. T. P. BEDDOES: There are two main divisions into which these products fall—namely, Class A, the so-called quack remedies lavishly advertised to the public; and Class B, which are first-rate products advertised to the profession only—and they may be discussed from different standpoints. It is necessary, however, at the outset to draw a very marked distinction between the two classes.

Class A.—What medical men think is that, whether quack and secret remedies are good, bad, or indifferent, they are an evil, because they are held forth to the public, by lavish advertisement, to be cures for various ailments which should properly be treated by medical men. Those that are made without skill are less objectionable than those made with great skill, because the former, after having attracted the attention of the public, would be less likely to obtain any reputation of being successful remedies.

Class B.—Chemicals, and preparations intended for use by medical men in their practice, which have been worked up to be first-class products, should be a help to medical men rather than the reverse, because they are intended to supply them with the best representatives of the drugs they wish to use.

Instances of these could be multiplied *ad infinitum*, but it may be mentioned that except for the Registered Trade Marks Acts the medical profession would be without some of their most valued remedies. Only by this protection could the introducers be repaid for the labour and expense of the manufacture and introduction to the profession.

¹ Adjourned from February 1, 1910.

The *British Medical Journal* attacks chiefly Class A, and only indirectly Class B, and then only to a very limited extent. The British Pharmaceutical Codex, in attacking Proprietary Remedies, deals only with Class B, and leaves absolutely alone Class A. It is noticeable that all the footnotes and new nomenclature in the Codex refer to substances and preparations which have been introduced for the use of medical men and which have the advantages claimed for Class B. The apparent object of these notes was to suggest a substitution for the original article on which the reputation of the product has been made.

The appeal which has been made to medical men to abstain from prescribing products under their trade names is only an extension of this attempt at substitution. It is not contended that the substitute is better than the original, but only that the profits arising from the sale of the latter go to the man who introduced it to the profession. The method of direct or indirect substitution may appeal to those chemists who prefer to buy at the lowest procurable price; the only possible result would be to drive the trade into the hands of large firms and those who sell cheapest, to the confusion of the prescriber, the enslaving of the pharmacist, and the detriment of the public. The result of this attempt at substitution produced a curious and impossible situation. Soon after the publication of the Codex wholesale houses were asked to supply some of the products under the British Pharmaceutical Codex titles, with the result that the orders were passed on to the respective original manufacturers of the products, who alone prepared the articles. The manufacturer naturally refused to sell his products under the new name, but was ready to supply it under his registered trade mark; in fact the British Pharmaceutical Codex product, as distinct from the original product, was unobtainable.

A medical man wishes to prescribe, say, a remedy for rheumatism which he knows to be of established repute in this disease, but he is adjured not to prescribe it under its registered trade name (which is the only way of securing the supply of the preparation he has in his mind), but to order it under its so-called chemical equivalent. His patient in that case may not get the preparation which he intended, but any variety of the particular chemical which a chemist may be able to procure, and this notwithstanding the fact that very slight alterations in the chemical and physical constants of preparations of this nature may account for the presence of dangerous impurities. Will the doctor obtain any advantage by taking this course? No! rather the reverse. The original manufacturer of proprietary products must of

necessity make his own interests identical with those of the medical profession. If, when introducing a product to the medical profession, he misrepresents it, his chance of retaining their confidence is gone; he would have small opportunity of repeating the offence. It is to his interest to only introduce products which he believes will be of general utility to the profession, and, when practice proves his statements to be correct, he has simply to keep his product at the highest state of perfection to retain general confidence.

A further point in the legal aspect is the question of copyright. The Pharmacopœia Committee of the General Medical Council reported on May 28, 1908, as follows: "The attention of the Committee having been called to a recent publication which appeared to them to infringe the Council's statutory rights in the Pharmacopœia, the opinion of counsel was taken on the subject. The opinion confirmed the view taken by the Committee, and suggested the proper steps to be taken for the Council's protection. Acting on the legal advice thus obtained, representations were made to the publishers, with the result that the present issue of the publication has been stopped. Guarantees have also been furnished that future issues shall not be open to the like objection." The Pharmacopœia Committee could not have done more to maintain their copyright and to protect public interests, because the book was issued by subscription and distributed before the Council could obtain legal advice—one of the most subtle forms of evasion of copyright.

The Codex has confused prescribers, was pernicious to the public, and it violated the first principle of literary copyright. The General Medical Council have earned the gratitude of both students and practitioners of medicine, as well as of pharmacy, for what they have done. For there is every reason to expect that the publishers of the Codex in future will avoid errors due more to want of thought than to want of knowledge.

Mr. F. W. GAMBLE said the paper read at the previous meeting by Professor Dixon was a very interesting one. He (the speaker) did not know why the discussion should take the form it had in the opening speech that evening—namely, an attack on the Codex. Professor Dixon might have referred to the Codex, but it was not more than a passing reference; the paper treated the subject very widely, and in such an admirable way as to avoid controversial matter.

With the general principles outlined he believed every pharmacist was in practical agreement. He was glad to find Professor Dixon

drawing the necessary distinction between such medicines as were advertised to the public and the goods which were advertised and recommended to the medical man. The pharmacist had no sympathy with the former; in acting as a distributor of these, every medical man knew he was an unwilling agent, but the distribution of such medicines had become an unavoidable part of the pharmacist's business, no matter where his business might be situated.

He (the speaker) believed the trade was a harmful one, and that efforts should be made to stop it. He hoped the discussion would lead to the adoption of some practical remedy for improving the present state of affairs. In America the declaration of the formulæ of proprietary medicines was compulsory, and he believed it was required by law in many Continental countries. That was surely a simple requirement, which, if adopted in England, would work great improvement. It would be some protection of the public against themselves, in that it would show them the character of the material they were taking; and it would enlighten some medical men who might otherwise prescribe a patent medicine without knowing what it contained.

The impression which Mr. Beddoes had conveyed concerning the Codex was not the correct one. That speaker inferred that there was an antagonism, or some outstanding difference of opinion, between the Pharmaceutical Society, or the Council of that Society, and the General Medical Council. That, however, was not the case. The relations between those two bodies were entirely amicable on that subject. When the next Codex was issued, it would accord with the wishes of the General Medical Council. As to whether the Codex had been the cause of great harm to medicine, there was room for an opinion vastly different from that expressed by Mr. Beddoes, and he would be inclined to leave it to the opinions of the gentlemen present as to whether that was the case. He had heard equally forcible expressions, but to the contrary effect.

Dr. J. GRAY DUNCANSON said he thought the paper a very good and practical one. The sale of such remedies, especially the secret remedies, was, in his opinion, a great and dangerous evil in this country; and it was, to a certain extent, a slur upon the medical profession. The people were very apt to take things for granted in the absence of denial, and it is only recently, in this country at all events, that a proper scientific attempt has been made to expose the constitution of those

nostrums. The press teems with advertisements of the most fraudulent and lying nature, which are apt to mislead very considerably; there was also a more subtle method sometimes adopted, which he took the opportunity of strongly denouncing. He referred to the practice of getting certain substances written up by members of their own profession, and then pamphlets or reprints of articles were distributed broadcast to the profession "with the author's compliments." To the British Medical Association they owed a debt of gratitude for a small book which it had recently published, entitled "Secret Remedies." He would advise every medical man to procure a copy at once, and it should be widely distributed throughout the land; if that were done, it would prove a powerful weapon in stamping out the evil. The people of this country were not fools, they merely wanted educating; they were being preyed upon by those who vended such things, and as soon as this was realized the demand for quack remedies would cease; he thanked Dr. Dixon particularly for the clear manner in which he had brought forward the legal aspect of the question.

Dr. WILLIAM MARTIN wrote, in a letter read by Dr. J. Gray Duncanson: "The subject is, as you say, one of great importance, and I am sure you could have invited no one better qualified than Professor W. E. Dixon to present it adequately to the meeting. I can see no good in the 'patent-medicine' method of foisting remedies on the public, and believe that legislation might be obtained that would substantially lessen or even stamp out the trade, if it were not that official discouragement would probably block the zeal of anyone who sought to do away with so fruitful a source of revenue; for the duties paid amount to a substantial sum every year, and it would be difficult to produce an imposing number of instances where danger to the public interest could be specifically proved—a line of argument that alone, in my opinion, would have any effect on Parliament. The enormous growth of patent medicines, with all the blatant and unscrupulous appeals to the populace through the advertisement columns of the press—lay, clerical, and pseudo-scientific—has become nothing less than a monstrous evil; but then you know the hackneyed Latin phrase, *populus vult decipi*. I consider, however, that within certain limits there is a distinct and legitimate field for the exercise of the 'protection' afforded by 'registered' names, and the output of what are generally known as proprietary preparations. You know, as well as I do, that manufacturers of medicinal products, like all other manufacturers, vary almost infinitely

in their sense of responsibility to their work, and if a maker, by scrupulous attention to every detail, from the selection of the crude drug up to the finished product, or by the application in certain cases of improved or special methods of manufacture, feels that he is able to put before the *medical* public a better product than the average, I do not see why he should not use legitimate means to reap the commercial advantage to be derived from his exercise of greater knowledge, skill, and care in his work; and the use of a 'registered' name enables him to do this in a way that frequently would not otherwise be practicable. There is here no disadvantage to the public because the appeal would be to the medical man, who is competent to judge of the matter. The case is far otherwise when manufacturers, whether using 'registered' names or not, put up their goods in a style that appeals to the lay user direct, and so tends to foster the practice of self-medication, and this is done nowadays to a larger extent than it ought to be, and is a subtle principle of business that directly traverses the province of medical men, whether viewing their interests from the professional or material point of view, and, worse than that, is as real a danger to the public as the use of 'patent' medicines pure and simple. For the vast majority of patent medicines, after all, are harmless; the damage is mainly to the pocket of those whose credulity is imposed upon; but in the type of business I allude to, drugs of dangerous potency find their way into the domestic medicine chest without any reference whatever to the medical profession. In regard to patent medicines advertised directly and openly to the public, I think that much of the evil might be overcome if it were required by law under sufficient penalty, as I fancy has been already done in some of our Australasian Colonies, that all medicinal preparations offered to the public should bear on the label a *full* statement in English of the contents of the bottle. The 'man in the street' would then soon find out that the use of the many much-advertised patent medicines forms an extraordinarily extravagant way of treating his ailments."

Mr. W. J. UGLOW WOOLCOCK desired, as a visitor, to give the meeting a little enlightenment on the book which had been referred to in somewhat scathing terms. It was properly named the "British Pharmaceutical Codex," not the "Pharmacopœial Codex." A closer acquaintance with the book would teach one that. He would also like to say why the book was published. In a subsidiary way it gave the composition of certain substances which had been known under trade names; and that object was not inconsistent with the main purpose of

the book, which was to compile a list of substances used in medicine. There were a large number of substances in common use which were not included in the "Pharmacopœia," for one or two reasons: either they were so old that the more advanced section of the medical profession had discarded them, though they were still in use among older practitioners; or that the "Pharmacopœia" was published at such long intervals that there must be many substances which had come into use in the last ten years, for instance, which should be in the book. The object of the Pharmaceutical Society in publishing such a book was that it should form a compendium of medicines; and they included in it all the drugs and chemicals which were at all in use, and felt also that they should include some of the drugs and chemicals recently introduced under their Latin names. Tempting though the subject was, he did not propose to enter into a discussion as to the division of secret remedies. Dr. Dixon admitted there were remedies of extreme value which had been patented, and those the medical man, as well as the pharmacist, required to know something about; and there were manufacturers who willingly supplied information about their drugs, and had supplied it in the form which had been presented to pharmacists and the medical profession. He supported the statement of Mr. Gamble as to the amicable relationship between the General Medical Council and the Council of the Pharmaceutical Society; it was idle to talk of an injunction or anything of the kind. The whole of the first edition of the "Codex" was sold out within six months, so that in case the impression should have been left on the minds of those present that at No. 17, Bloomsbury Square there was a large stock of the "Codex" which could not be sold, he wished to say that was quite a mistaken idea. He thanked the Society for the permission to address the meeting.

Mr. E. F. HARRISON said he was very glad to know that the Section was devoting two evenings to the discussion of the matter, as it was one of very great importance, and one on which the attitude of the medical profession would very largely influence the public. He thought Dr. Dixon had made rather too light of the importance of the ordinary quack medicines from the medical man's standpoint. People often procured quack medicines when they would not go to consult a medical practitioner; and the medical man had many means of properly influencing the public, such as the way mentioned by Dr. Duncanson, by letting them see the composition of the quack medicines they took. It was true that patent medicines often contained but little of what was

harmful; but he wished to point out that it was not necessary for a patent medicine to contain dangerous drugs for it to be a dangerous preparation. In the little book referred to there were two or three preparations which were advertised very largely for the cure of consumption and for the cure of cancer, and there was one, called "Tuberculozyne," for which the charge was £2 10s. for two bottles; the cost was 3d., and it consisted practically of coloured water. That was advertised in the most persistent way, so that many people in a more or less advanced stage of consumption were induced to go on taking it, by carefully drawn-up letters, sent at short intervals, asking the purchasers not to be discouraged, as patients got worse first before the cure set in. Consequently people did not go to a medical man until it was too late to do anything except make their remaining days more tolerable. Much the same sort of thing was done in the case of cancer. In seeking to stop such things there were the greatest difficulties to face, because some of the preparations showed skill and research, and had value; and for those the individuals in question could claim some particular right, a right of which no fair-minded person would wish to deprive them, or of any emoluments they could get from them; between those and the fraudulent quack remedies, there were all gradations. When an attack was made upon proprietary medicines, it was found that the proprietors of the good ones rallied to the support of the proprietors of the bad ones. That was seen three or four years ago, when legislation was proposed in New Zealand which would have had the effect of keeping out some of the patent medicines sent from this country. A meeting was held of the Chemical Section of the London Chamber of Commerce; and there sat, side by side, proprietors of desirable and undesirable proprietary articles, and they made common cause to defeat the regulations. Thus it should be made clear, in attempts to deal with the matter, that no attack was intended on the bona-fide proprietary article; and if insistence were made on a declaration of the composition, at least as regards the main ingredients, he did not think there would be much objection on their part. Every detail need not be given, so that any particular skill in the combination would receive its due credit. The medicines which would be injured by such a provision would be those which derived some part of their revenue from quackery. He hoped some practical result would arise, either as the outcome of the present meeting or of the issue of the little book referred to; and that there would be some legislative enactment. In any attack on patent medicines it was best to avoid doubtful cases at first; but in the case of such a

thing as "Tuberculozyne" one should be absolutely unsparing. Many of them were run as syndicates, and if attacked they might fight; but, as they must know publicity was the last thing they should have, for their own sakes they might take the wiser course. An enormous amount of matter emerged which might be profitably discussed, and he felt much indebted for the permission to speak on the subject.

The PRESIDENT (Professor Cushny) said that some of the plans suggested to combat the nostrum evil reminded him of what had happened in the United States in the last ten years. Ten years ago perhaps no country in the world had the benefit of so much gratuitous advice as to treatment given to both the medical profession and the general public. Day by day the medical man was deluged with advertisements which were also in the daily press and in broadsheets of every kind. About six years ago the matter was taken up by the American Medical Association at the instance of its very able and energetic editor, Dr. Simmons, and a committee of experts in pharmacology and pharmacy had been formed who had separated the wheat from the chaff in proprietary medicines and had organized an attack upon the unsatisfactory products. That attack had very remarkable results with regard to the use of patent remedies in the United States, and particularly in regard to the way in which they were employed by the medical profession, for the latter were the chief people who were previously exploited by the patent-remedy manufacturers and the quack-remedy manufacturers. And the result appeared to have been very satisfactory, for the medical profession had risen to the realization that they were being exploited. The success of the movement was shown by the loud protests raised by the manufacturers of the quack remedies. The British Medical Association had not taken quite the same position as the American Medical Association. The latter began by issuing a small list of approved remedies—not of disapproved remedies—and he was not sure that the American method was not the better one. The list of approved remedies, which were required to be kept uniform and which could only be advertised in a proper way, was issued to the profession only, and the better-class manufacturer appeared to regard the action of the Association as beneficial to his work.

Professor DIXON, in reply, said it had been a matter of regret to him that more speakers had not suggested a means of dealing with some of the difficulties in connexion with the matter. When to-day's discussion

started he was for a time alarmed that it was going to be entirely off the line. In an unguarded moment he mentioned in his paper the word "Codex," and that appeared to have acted as a sort of red rag to Mr. Beddoes. As Mr. Beddoes could not have appreciated what he (Dr. Dixon) said about proprietary remedies and trade names, he would read again one sentence in the paper: "Proprietary medicines not of a secret nature are those with which the medical profession are mainly concerned, and, for the introduction of some of these, medical men and the public are indebted to some of the great wholesale firms which have fully-equipped pharmacological laboratories," and then he gave a list of them. If, through the ingenuity of a firm or an individual, a new substance was formed which was found to be of value in medicine, one could not do too much to protect that substance for the benefit of the discoverer for a reasonable time. If new, the man would probably protect his substance by letters patent, and give it some characteristic name, and by this means he enjoyed protection for fourteen or twenty-one years. But what did these fancy names mean when the substance was not patented? They generally signified that some more or less well-known chemical substance was being foisted on the medical profession under a trade or protected name. Acetyl salicylic acid was an example of one such substance, and a wholesale house gave it the name of "aspirin." Five or six years later other wholesale houses, finding aspirin was being largely prescribed, gave the same chemical substance other fancy names and sold it under those names, so that now seven or eight names existed for the same substance. It was obviously most desirable to get rid of the many fancy names for the same substance. He regretted that speakers did not devote more attention to the means for remedying the state of affairs now existing. In his paper he suggested three possible means. One of them Mr. Harrison had referred to—namely, that the composition of proprietary remedies should be labelled on the box or bottle. That he regarded as the most essential one, but one which they would have great difficulty in carrying out, because proprietors would employ all their resources in fighting such requirements, and in this they would be assisted by the Press. The other two suggestions were to apply the Merchandise Marks Act and to make the Food and Drugs Act applicable to proprietary medicines. At present the latter Act only applied to the chemist, who could be prosecuted if he sold a substance which was not pure.

The Treatment of Infective Endocarditis.

By JOHN COWAN, M.D.

It has been my fortune in the last few years to encounter a large number of cases of acute endocarditis. They have been, of course, of very varied nature: some of them occurred in cases of acute rheumatism and chorea, and were recognized by their physical signs rather than by the occurrence of cardiac symptoms; while others were only diagnosed post mortem as the pathological explanation of symptoms affecting organs other than the heart. With the treatment of such cases I do not propose to deal in this paper. In the first group, recovery is the rule; in the second, treatment is without avail. But there remains for consideration a large number of cases where the cardiac infection is severe but sub-acute, where it is duly recognized, and where sufficient time elapses to allow any line of treatment to be carried into execution.

The diagnosis of acute endocarditis is often extremely difficult, particularly in hospital patients with unknown antecedents and a cardiac lesion of unknown duration. Dr. Horder has suggested that the occurrence of emboli and the isolation of organisms from the blood are almost conclusive evidence of an infective endocarditis; but this definition excludes practically all the cases of rheumatic origin, though their course is not infrequently malignant, as it is but rarely that the diplococcus has been obtained in this way. I am inclined to lay particular stress on variations in the cardiac sounds or murmurs, and in the size of the hearts, *occurring under observation*, as indicative of a lesion that is progressive and so acute; and on the presence of pericarditis as signifying an acute cardiac lesion elsewhere. Recently, too, I have found further assistance in the routine use of the polygraph.

We are at present accustomed to consider that the conduction of stimuli from auricle to ventricle takes place along the auriculo-ventricular bundle; and that in certain cases ectropic stimuli may arise in the same situation. Defects in conduction and extra-systoles may of course occur from other causes than changes in the bundle, notably from vagus influences; but the close proximity of the bundle to the anterior cusp of the mitral valve and the non-coronary cusp of the aortic renders it extremely liable to be involved in inflammatory lesions affecting these valves; and, as a matter of fact, it is well known to be often implicated.

In acute heart disease temporary or permanent alterations in conductivity are not infrequently observed to occur, and also changes in the site of origin of the stimulus, and *their occurrence under observation* is at any rate very suggestive of the presence of an inflammatory lesion on the affected valves.

A woman, aged 36, was recently admitted into my wards suffering from arthritis and fever of two weeks' duration. The left heart was enlarged and a mitral murmur was present. The pulse was infrequent, but cervical tracings showed that the auricles contracted much more frequently than the ventricles, defective conductivity causing the ventricles to respond to only every second auricular contraction. Six days later the *a-c* interval was normal and the pulse more frequent, but three days afterwards conduction again became defective, and this persisted for another six days (figs. 1, 2, 3, 4). The combination of fever, mitral incompetency, and temporary defects in conduction is, I think, very suggestive of an acute process within the heart. I do not think that these defects in conduction are at all rare. At any rate, I have found five examples in my own wards within the last twelve months.

The next tracing was obtained from a man, aged 24, who was suffering from symptoms which were due to an embolus in the left middle cerebral artery. The close proximity of *a* and *c* in the jugular tracing (fig. 5), even though the sequence is regular, makes it probable that the stimulus did not arise at its normal site, but at some point in the primitive tissue, not far distant from the ventricles; perhaps from involvement of the auriculo-ventricular node in the same process that had attacked the mitral valve. This is the second case of the kind which I have had under observation.

The cases whose treatment I propose to consider are those of more or less chronic or sub-acute character associated with symptoms, in some cases of cardiac distress, in others of a general infection. Their aetiology is very varied, and many micro-organisms may be causal; but from the clinical point of view they may be divided into three groups: (1) those in which bacteria have been isolated from the blood; (2) those which are apparently of rheumatic origin; (3) those whose course is malignant, though blood cultures are negative, and which may or may not be of rheumatic origin.

(1) My personal experience of blood cultures is comparatively small, but my results are by no means as satisfactory as some of those which have been published, and it has been the exception to isolate

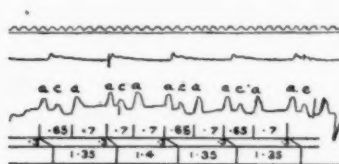


FIG. 1.

January 13, 1910: Partial heart-block in acute rheumatism. Every second auricular contraction fails to produce a ventricular contraction. Time marker = 0.1 sec.

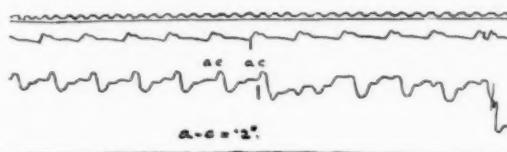


FIG. 2.

January 19, 1910: The heart-block has disappeared and the $a-c$ interval is of normal duration.

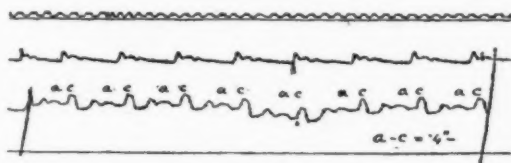


FIG. 3.

January 24, 1910: Recurrence of defective conduction; $a-c$ interval is prolonged.

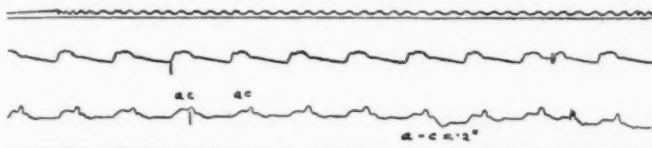


FIG. 4.

January 28, 1910: The heart-block has again disappeared.

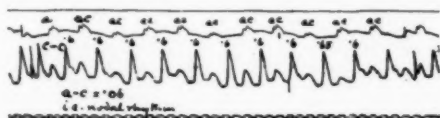


FIG. 5.

Infective endocarditis.

the bacteria during life, even though they have been easily obtained post mortem. It seems reasonable, however, whenever a definite organism has been isolated, to administer the corresponding antiserum, even though one must, as a rule, employ a polyvalent serum, and not one specially prepared for each specific case. But I hope that ere long the bacteriologists will enable us, by some agglutinative or other test, to choose for each individual case a serum which is specific rather than generic for the particular micro-organism which is concerned. Vaccine treatment has been advocated in this type of case, but the results hitherto published have been disappointing, and I am extremely doubtful if good results will ever be obtained by this means. The production of antibodies, which is the aim of vaccine treatment, takes place in disease in response to the presence of bacteria or their products, and may fail to be sufficient if the infection is local and absorption minimal, or if the infection is intense and the defensive processes are overpowered. Of all the conceivable sites of infection, the endocardium is surely that most certain to produce a general intoxication, and I fail to comprehend how in these general "arterial" septicæmias an additional dose of the poison can be of benefit to the patient.

(2) In the second group of cases, those apparently of rheumatic origin, the question of salicylate treatment has to be considered. Is the occurrence of endocarditis in acute rheumatism a contra-indication of their use, or a reason for their administration in larger doses? To answer this is difficult. I am accustomed to use alkalis in combination with salicylate of soda, and I have often given 120 gr. to 180 gr. of the latter salt in the twenty-four hours; but my results with the larger doses have taught me to watch these patients very carefully, particularly if they are of middle age with a high blood-pressure and defective elimination. Cerebral symptoms have been uncommon, and have never given rise to any special anxiety, but I have sometimes thought that the cardiac action was weakened by their use, and have cut down the dose with apparent benefit to the patient. I never give large doses in cases with pericarditis or dilatation of the heart, but rather from the slighter evidences of their interference with cardiac action than from the occurrence of any serious accident. Arthritis is not an uncommon symptom in streptococcic infections, and many of the chronic cases are at first given salicylates, the true nature of the disease being suspected on the failure of the salicylates to influence the symptoms; but I am rather sceptical that failure to react to salicylate treatment indicates a non-rheumatic infection. Acute rheumatism may run a malignant

course in spite of salicylate treatment, which, indeed, produces its most striking effects in the slighter cases, and the presence of endocarditis is surely an indication of the severity of the attack.

(3) The third group of cases is that which occasions the greatest perplexity from the therapeutic point of view. Salicylate treatment is probably always instituted at first from a mistaken (?) diagnosis, but fails; and serum treatment is negated by the non-recognition of the causal bacteria. I am by no means opposed to serum treatment, and have indeed a full belief in its value in proper cases, but I do not believe in the haphazard administration of such powerful substances, and consider that their use should be confined to cases where the infection is definitely recognized, and a specific antibody can be procured.

We are all, I presume, agreed that in these cases complete rest in bed, fresh air and good nursing are necessary, and that food should be supplied in as full a measure as is possible, and in any form that can be utilized by the particular patient; solid food may be well digested even by fevered patients. But how are we to act in other directions beyond simply treating symptoms as they arise? Is there no method of directly combating the infection? We probably do not fully realize the fundamental differences between bacterial and protozoal infections, but the success of drug treatment in malaria and in syphilis makes it possible that in time an antidote to the pyogenic infections may be discovered. A survey of the literature, however, and a consideration of the numbers of drugs which have been recommended in these cases makes it very evident that no antidote has as yet been found.

Collargol is a drug which was introduced by Credé in 1895, and has been largely employed in the treatment of local and general infections of various kinds. It has slight bactericidal action, but it inhibits the growth of bacteria upon nutritive media. Its administration is followed by a leucocytosis, and it is said to possess powerful catalytic properties and to "provoke, increase, and quicken the processes of oxidation within the organism." It has been used in acute endocarditis by many observers (Wenckebach, Klotz, Sängner, Cohen, Netter, Müller, Legendre, Edsall, Hochheisen, Kornfeld, Ricci, Widal, Faure-Beaulieu), who have, on the whole, reported favourably. I have used it in five cases of acute endocarditis.

W. N., age 19, miner, was admitted on November 7, 1908, complaining of feverish symptoms of a week's duration (fig. 6). He fell ill on October 31, 1908, the initial symptoms being sickness and chilliness with frequent shivers, and he was forced to go to bed early in the evening; when in bed he felt hot

and cold alternately, and sweated profusely. These symptoms continued, and on November 3 almost every joint in his body became painful. The pain was very severe at first, but had subsided to a great extent next day. On the morning of admission he experienced severe pain over the front of his left chest, but it passed off after a few hours. He had previously suffered from two similar attacks: one at the age of 12, when the arthritis was severe and confined him to bed for four weeks; and again at the age of 15, when he was in bed for three weeks. But with these exceptions he had been healthy, and he had worked as a "hewer" in the pits without discomfort for the past four years. The family history was unimportant. On admission he was found to be a well-nourished, well-built man, and he lay easily in bed without discomfort, although many of the small joints of the fingers and toes were swollen and tender, and both knee-joints were distended with fluid. His skin was moist; the tongue was heavily furred, and the fauces were congested. The abdomen was slightly distended, but the stools were normal. The lungs were clear. The urine contained a trace of albumin. The apex impulse was distinct but somewhat diffuse and only slightly displaced. Systolic murmurs were audible all over the precordial region. At the apex the murmur was loud and long, replacing and running out of the first sound, and being well conducted into the axilla: the second sound was inaudible. The second sound in the pulmonic region was emphatic, and was followed by a short, soft, diastolic murmur. The systolic murmur was less loud at the aortic cartilage, and was conducted into the neck: the second sound here was indistinct. Soft superficial friction sounds were audible at the xiphoid cartilage. For about a month after admission patient's condition remained critical. The arthritis rapidly subsided, the tongue cleaned, and his digestion improved, so that he could take a fair amount of nourishment; but the fever persisted, and he became very white and exhausted. There was some congestion at the bases of the lungs, and cardiac weakness became extreme and accompanied by dilatation of the right heart. There was but little pericardial effusion and the friction soon disappeared. Towards the end of December, however, some improvement was apparent, both in his general condition and in the cardiac strength. The pulse became of better quality and less frequent, and the diastolic murmur lengthened out and assumed a definite aortic distribution. But it was late on in January, 1909, before the dilatation of the right heart had lessened, and the middle of March before he was able to sit up in a chair. When he left on May 1, 1909, he was fairly well, being able to go about the ward without any discomfort, but he was easily tired and made breathless by even slight exertion. There was never any definite evidence of embolism; but he complained at the end of November of pains in his left side, and in April in his right pectoral muscles, which may have been due to such a cause. Bacteriological examination of the blood on November 19, 1908, was negative. His subsequent history was short. After his return home he remained in fairly good health until the end of July, when he "caught a chill," and he died at home three weeks later.

D. L., aged 20, fireman, was admitted on May 21, 1909, complaining of pains in his joints of six days' duration (fig. 7). His previous health had, on the whole, been good, save for an attack of measles as a child, and pneumonia at the age of 16. He was, however, liable to tonsillitis until he was 13 years of age, but subsequently it had ceased to trouble him. Three weeks before admission, however, his tonsils became again acutely inflamed, and he was in bed for a week. As he then had quite recovered, he resumed his work, which exposed him to the weather, as he was on a night shift. On the evening of May 16 he began to feel pains in the "small of his back;" on the evening of May 18 his shoulders began to be painful, and on May 19 the hips were affected and he was compelled to go to bed. Next day many more joints were implicated, and he sweated profusely. On admission to hospital he was found to be a well-nourished man of good physique. His face was flushed and he was sweating profusely, and evidently suffering considerable pain. The tongue was furred, the mouth foul, and both tonsils were enlarged and slightly inflamed. Both knees and ankles, the wrists, and many of the phalangeal, metacarpal-phalangeal, and metatarsal-phalangeal joints were swollen and tender. There was a considerable amount of fluid in the right knee-joint. The apex impulse was faint and was not displaced. On auscultation the sounds were pure, but the first sound was extremely short and indistinct and somewhat distant. The pulse was regular and fairly full, but soft and frequent. The other viscera seemed normal, save for a little catarrh in the lungs. The arthritis rapidly became less acute, but persisted in one or other joint until the end of June. There was a considerable effusion into the left knee-joint after admission. The fluid in the right knee-joint, which was examined on May 23, was very viscid, straw-coloured, and almost purulent. The films were mainly composed of well-preserved neutrophil polymorphs, with a few lymphocytes and epithelial cells. No bacteria were seen. The tonsillitis persisted for some time after admission, but the inflammation had subsided in the first week of June. In July, however, it returned and persisted for a week, and again in the last week of November. His general condition slowly but steadily improved after admission, and he was always able to take an adequate supply of nourishment. Bacteriological examinations of the blood on May 7, 1909, and July 11, 1909, were negative. The cardiac condition never gave rise to any anxiety from the symptomatic point of view, though the auscultatory phenomena indicated grave myocardial weakness. The first sound remained short and distant but pure for about a fortnight, after which it began to lengthen from the addition of a systolic murmur of mitral distribution. Towards the end of July a short, soft diastolic murmur became audible in the third left space about an inch from the sternal margin, succeeding an emphatic second sound. The pulse remained frequent at first, but reached normal limits at the end of July. He was allowed out of bed in the commencement of October, and when dismissed home on January 24, 1910, was able to take a fair amount of exercise and to walk upstairs without any evident discomfort. The heart was slightly enlarged, and the aortic and mitral murmurs persisted.

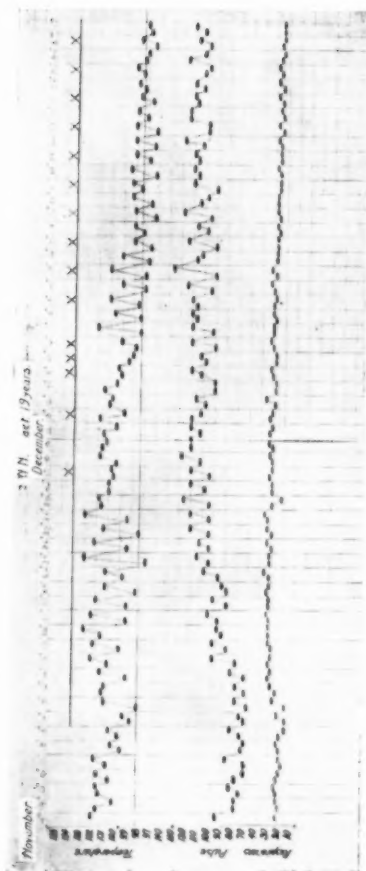


FIG. 6.—Improvement ensued during the period of injections.

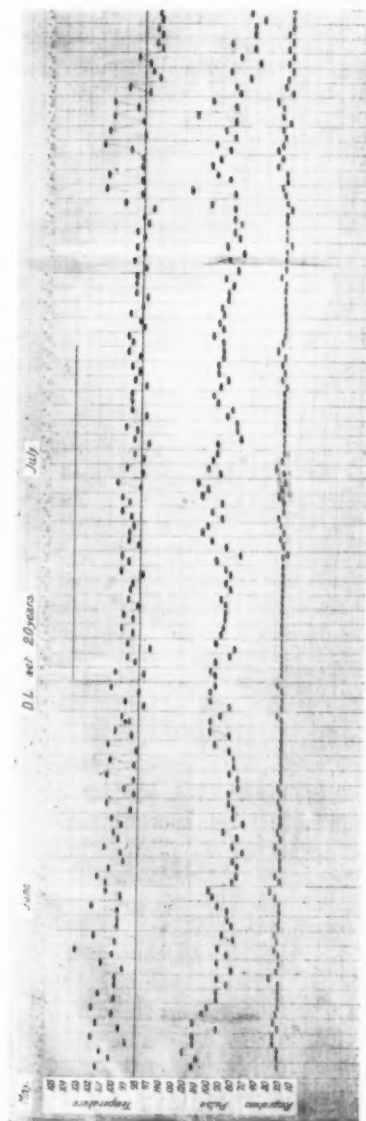


FIG. 7.—Improvement, which had already begun, continued more rapidly during the period of inunction.

In these figures the upper line indicates the periods of inunction of collargol.
The x x x indicate intravenous injections of collargol.

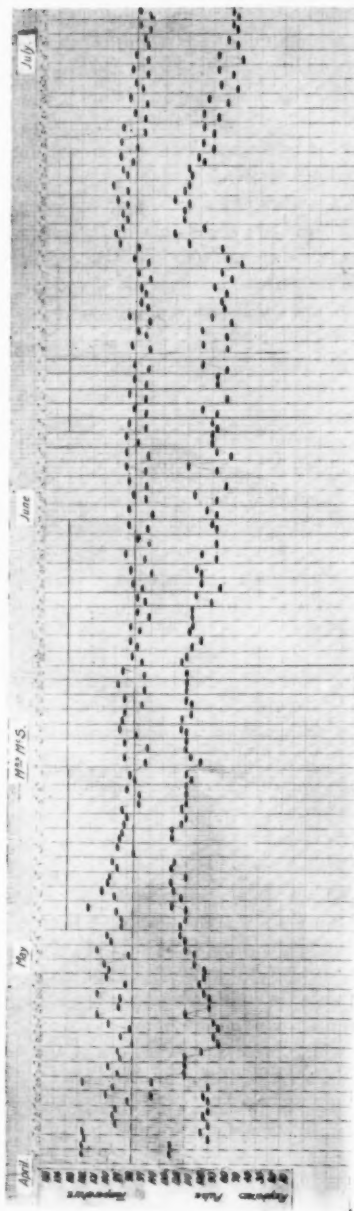


FIG. 8.—The cardiac symptoms were serious at the time when inunction was begun and steadily improved afterwards.

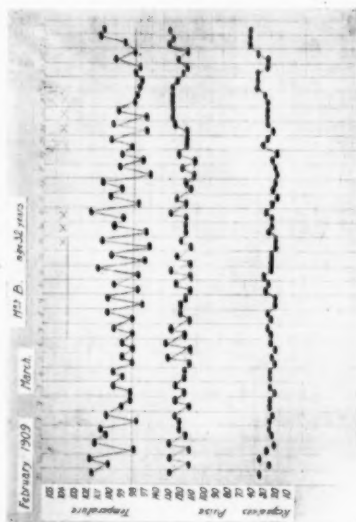


FIG. 9.—No improvement.

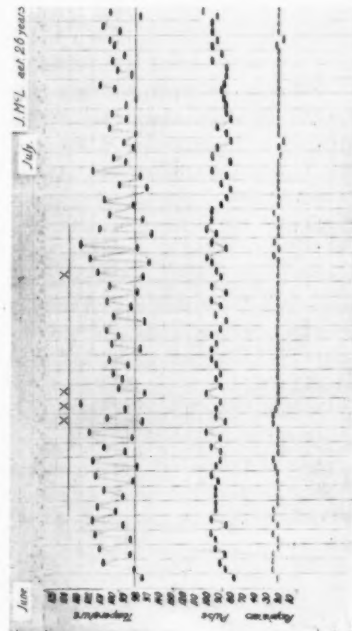


FIG. 10.—No improvement.

B. M., aged 14 (fig. 8).¹ The illness commenced with a severe attack of tonsillitis in the first week of April, 1909, accompanied by transitory pains, without objective signs, in the right hip and knee. In the course of a fortnight both the tonsillitis and the pains had subsided, but the pyrexia persisted and the pulse-rate continued frequent, and a mitral murmur made its appearance. When I saw her for the first time, with Dr. Young, in the beginning of May, she seemed to be well nourished, but looked white and ill. Cardiac pulsation was widespread in many interspaces and on both sides of the sternum, and the right heart (not the left) was evidently dilated. A systolic murmur was audible everywhere, replacing and running out of the first sound and occupying almost the whole of the short pause; it was heard best at the apex and was well conducted into the axilla. The second pulmonic sound was emphatic. The pulse was regular, frequent, and fairly full, but was easily obliterated. The family history was unimportant. The child had had no serious illnesses before the present one, and the mitral lesion was known to be of recent development, and, in the absence of other visceral lesions, was considered to be the cause of the fever. Blood-cultures on May 4, 1909, were sterile. Progress was continuously in the right direction, but tedious and slow. Pyrexia persisted more or less until the second week of June, and the pulse-rate only reached normal in the beginning of July. The dilatation of the right heart subsided, and in September she was able to take short walks without discomfort. In November she contracted another tonsillitis, but it soon passed off and the heart did not seem to be again involved. At the present time she is in good health and without any cardiac symptoms, though the mitral murmur persists and the left heart is slightly enlarged. Her growth has been but little interfered with.

Mrs. B., aged 32, was admitted on February 25, 1909, complaining of pains in her joints for the past two months (fig. 9). She stated that she had always been a healthy woman until the commencement of her present illness, with the exception of a slight attack similar to the present one two years before, which, however, only lasted for a week. She had been pregnant five times, but the last three terminated before full time; one of her children died when aged $4\frac{1}{2}$ from whooping-cough. Her labours were always easy and were followed by a normal convalescence. Her present illness commenced with pain and swelling in the right ankle, which forced her to go to bed at once; two days later the right knee became affected, and within the week the arthritis became general; she was feverish at this time and slept badly. She has never been well since the commencement of her illness, and she has been confined to bed throughout, at first mainly on account of the pains in her joints, but latterly from weakness, as the pains, though still persisting, have lessened in intensity. On admission she was seen to be a badly nourished, anæmic woman of fair physique and good muscular development. There was a widespread eruption of erythema circinata all over the trunk. The right wrist-joint was slightly swollen and tender, but the other joints were normal. The tongue was slightly furred, but the throat

¹ The chart (fig. 8) is erroneously marked Mrs. McS.

was clean. The axillary glands were considerably enlarged, but were not notably tender; the glands in the groins were slightly enlarged. The edge of the spleen could be felt just below the costal margin. The lungs and abdominal viscera seemed normal, though the liver was slightly enlarged, and the urine contained a trace of albumin. The pulse was regular and frequent, but very quick. The cardiac pulsations were widely diffused, and the left heart was enlarged. At the apex the first sound was replaced and followed by a soft, blowing murmur, which was well conducted into the axilla; the second sound was double. The second pulmonic sound was emphatic, and was followed at the left sternal junction of the third and fourth costal cartilages by a short, soft whiff. For three weeks after admission there was but little change in the patient's condition. The arthritis soon subsided and did not recur, but the fever persisted, and, although she was able to take her food fairly well, her general condition on the whole deteriorated. Repeated examinations failed to reveal any lesions other than the cardiac murmurs and the glandular enlargements. Blood-cultures in the second week of March showed a small growth of *streptococci*. In the last week of March symptoms of increasing cardiac weakness became manifest; she suffered from attacks of breathlessness at night, and was frequently sick, and the liver became enlarged and tender. On March 28 she spat up some bloody sputa, and râles were noticed at the bases of the lungs. The skin became slightly yellow, and her general and cardiac weakness were extreme. She was removed home in a moribund condition and died in a few days. The duration of her illness was about three months.

J. M., clerk, aged 26, was admitted on June 5, 1909, complaining of weakness and of breathlessness on exertion of some months' duration (fig. 10). He stated that he had not been in good health for some years. His first illness was an attack of enteric fever, for which he was in hospital in Japan from October to December, 1903. When he was allowed out of bed after this attack, his feet became swollen, and he never regained his former strength. He was able, however, to work as a clerk and a ship's steward, though he found the latter work heavy, and he was engaged more or less continuously until July, 1908, when he was thrown out of employment by the failure of his firm. He stayed at home, in fairly comfortable circumstances, until he procured a new post as a clerk on February 19, 1909. In the early part of February he had caught cold, and he was feeling "run down" when he commenced his new work. This entailed a daily walk of four miles, and he felt exhausted at night, and also during the day when walking up a hill; but, though he steadily became weaker, he continued at his post until six weeks before admission, when he was compelled to go to bed. His cough was accompanied by a slight mucoid spit, and persisted from February until May. Since February he had had frequent attacks of shivering at night after getting home. They were fairly severe and lasted for fifteen or twenty minutes, and he felt hot after he got into bed, and often sweated profusely in the early mornings; the sweats have been less frequent since he has been confined to bed. About a fortnight before admission his right forearm and elbow became sore to the touch and on movement, and this

persisted for a few days. Four days ago his left hip and ankle became painful, and this still persisted. His family history was good. Prior to 1903 he had always been healthy, though he admitted on cross-examination that he had suffered from "growing pains," and one attack of tonsillitis in boyhood. He had also had an attack of gonorrhœa early in 1903. On admission he was found to be a thin, pale, badly nourished man with flabby muscles and little subcutaneous fat. The left hip and ankle were painful and tender, and there was a small tender area of induration on the back of the left forearm. The right calf was, he said, painful, but seemed normal on examination. There were several minute ecchymoses on the conjunctival surface of the right lower eyelid, and a fairly large one on the conjunctiva of the left bulb; while ophthalmoscopic examination revealed a medium-sized flame-shaped hæmorrhage on the course of the left superior temporal vein. The lungs were normal, but the liver was definitely, and the spleen probably, enlarged, though neither could be felt in the abdomen, and the urine contained a small amount of albumin. The tongue was clean and digestion apparently normal. On examination of the heart the impulse was found to be diffuse and badly sustained, but not displaced. A loud blowing murmur was audible all over the præcordium. It was heard best at the apex, where it replaced and followed the first sound right up to an indistinct distant second sound. The murmur was well heard in the axilla and at the left scapular angle. The second pulmonic sound was emphatic. The pulse was small and soft and regular. Progress after admission was slowly but continuously in the wrong direction. Many emboli occurred, mainly in the limbs, but once, at any rate, in the spleen, and twice in the kidneys. He had several faint turns of a few minutes' duration without obvious cause. The weakness increased, and after the middle of July œdema was more or less constantly present. There were occasional rigors, and generally profuse sweats at night. Towards the end the fever ran at lower levels. On August 25 a left hemiplegia occurred suddenly, and he died on August 29. Blood cultures on June 19, 1909, and August 12, 1909, were sterile. Post-mortem examination revealed an acute endocarditis of the mitral valve with many vegetations, some small and warty, and others large and luxuriant. Two emboli were found in the right middle cerebral arteries, and septic infarctions were present in the left kidney and spleen; from the latter the *pneumococcus* and *Bacillus coli communis* were isolated on culture.

The cases which I have reported represent all my experience with collargol in acute endocarditis. The first case was not, in my opinion, of rheumatic origin, and the result seemed to be due to the administration of the drug. I am unfortunately ignorant of the cause of death, but from the extent of the valvular flaws it was not unexpected. The second case was probably rheumatic, but was a severe and protracted infection with marked cardiac involvement. Collargol perhaps helped in the fortunate issue. In the third case I do not think that the

cause was rheumatic. The temperature chart seems to show that improvement was occurring at the time that collargol was first administered, but the cardiac symptoms were then extremely threatening, and their improvement dated from the commencement of its administration. In the fourth and fifth cases no effect was produced. In the last case I was disappointed with the result, as the case was essentially chronic; but the patient's general nutrition was extremely poor, and the anæmia was considerable.

I am confident that no harm resulted from the administration of the drug, and I consider that my experience warrants its further trial in the type of case which I have described.

DISCUSSION.

Dr. JAMES MACKENZIE said his experience of severe infective endocarditis was very small; he had generally only discovered the condition when the patient was dying, so that he had had no means of testing remedies for it. In the cases he had seen the chief symptom was violent fever, and he was not on the look-out for anything of the sort until an infarct occurred. Dr. Cowan had led the Section to infer that when in a febrile case he discovered a murmur, it was likely to mean that active changes were going on; but he (the speaker) did not think so. He had worked a good deal at hearts in febrile states, and he found in some cases dilatation of the heart occurred very readily, whereas on the subsidence of the fever there was no serious affection of the valves. One patient had been burned, and had a temperature of 107° F. After a consultation it was concluded that there was a malignant endocarditis. But in a week the temperature was normal and the patient recovered. That was fifteen years ago, and he is still in good health. He was glad to hear of the systematic examination of those febrile hearts for alterations of the rhythm. In that, he thought, they would get explanations of many obscure conditions. Ten years ago he made observations identical with Dr. Cowan's in a case of severe influenza, in which there were changes in the bundle and heart-block. The latter persisted only a few days, and the patient made a good recovery and was to-day sound and vigorous. He also had had a case which began with mild rheumatic fever, with a systolic mitral murmur, and a systolic aortic murmur developed afterwards, and the tracing of the jugular pulse made him suspect delayed conductivity. He put the patient on digitalis, and produced by that means heart-block. He had seen the same sort of thing arise in sub-acute cases; and one case of nodal rhythm shown on the screen was interesting. The President had produced that experimentally by means of aconitine; and Dr. Lewis had produced the same condition by ligation of the coronary artery. It meant that if one allowed for the pre-sphygmographic interval,

the time between the contraction of the heart and the opening of the aortic valves, the auricle and ventricle must have gone off together, and there must have been a starting of the contraction of the heart from a new centre; probably from the poisoning effect and irritation of some part of the node producing that result. Altogether, Dr. Cowan's paper was very suggestive and interesting.

Dr. SAINSBURY said he had come not to speak but to learn, as he happened to have a bad case at present under treatment, and he hoped he might hear some hint as to further treatment. In his own case he had tried both anti-serum and vaccine, but without good effect. He was glad to hear Dr. Cowan's protest against the use of a vaccine in cases of the kind. In using it himself he had yielded to the recommendations of the pathologist, not to his own convictions. For he did not see how one could rationally expect that an additional dose of a poison should give good results in cases of general infection such as these. Reverting to his own case, he had obtained the best results thus far from the use of quinine in 30-gr. to 40-gr. dose per diem, but unfortunately there had been a recrudescence of the pyrexia after some seven to ten days of normal, or practically normal, temperature. Creosotal, in doses of 15 gr. to 30 gr. every three or four hours, was entirely without effect, good or bad, and he was now proposing to revert to quinine, but by hypodermic administration.

Dr. ALEXANDER MORISON said it was important to have a definition of infective endocarditis. He had hoped to hear information concerning a more successful treatment of the condition. But Dr. Cowan's cases were not those which were conventionally placed in the category of infective endocarditis. Endocardial inflammation was of course known to be the result of an infection. The cases in which one failed to grow any organisms after repeated attempts were, however, in a different category from those where one could do so; and the cases where Dr. Cowan had used the remedy with apparent benefit were those in which no organism was isolated. He agreed that vaccination in septic cases had been quite futile; indeed, it was almost illogical to expect a good result therefrom: it was as reasonable as expecting to modify a small-pox attack by vaccination after the disease had begun. Vaccination in such cases might prove a preventive, but scarcely a curative measure. People who had some old lesion might conceivably be vaccinated with success with some polyvalent vaccine, and, as a consequence, a subsequent attack might not run into a septic form. But if they were to term rheumatic endocarditis infective in the same sense, then the cases in which one could grow the organism should be called "septic," so that the two might be differentiated. Rheumatic endocarditis, even of long standing, frequently got well. When cases did not get well quickly, he (the speaker) had the blood examined to see if it contained an organism; and if it did not, he usually found eventual recovery. But if an organism was found, even if the patient seemed fairly well, he was nevertheless doomed. He would not say cases never recovered, but he had never met with such, and regarded with scepticism reported recoveries.

Dr. NEWTON PITT said that his experience of the use of antistreptococcic serum, although sometimes good, had, on the whole, been evil. Taking thirty-nine cases in the last ten years, there were only two, which he published some years ago, in which recovery was apparently due to the serum. One was a boy, aged 8 or 10, who had infective endocarditis with a hectic temperature. He became profoundly ill, had albuminuria, with petechiæ and right hemiplegia, and at one time was apparently moribund. He was then given antistreptococcic serum regularly for some time; he finally recovered, and was in comparatively good health eighteen months afterwards. With these exceptions, he had not seen definitely beneficial results from the treatment. He recalled three or four in which the results were profoundly evil. One was that of a man with infective endocarditis from whose blood streptococci were cultivated. An antistreptococcic serum was prepared, and the patient became markedly worse as the result of the injections, and was himself convinced that he felt worse after each one. He developed a much more acute process, with septic emboli, and died. His end, in Dr. Pitt's opinion, was hastened by the treatment. In another case in which the endocarditis was not very acute, it seemed to light up as the result of the injections. In one or two others the temperature oscillations were increased. The general conclusion to be drawn from an inspection of a series of charts of cases so treated was that the result on the course of the disease was often inappreciable. He was, on the whole, inclined to think that the patient had an equally good or a better chance without the antistreptococcic serum. His experience of vaccines had been limited but unfavourable. There is already some evidence that we may possibly in the future be able to obtain some useful results from the intravenous injections of metals, such as arsenic and mercury, if they can be administered in a form not readily excreted by the liver or kidneys; he was not convinced that such medication would be useless for septicæmias when due to bacteria, as had been suggested. He had been struck by the fact that but in a very small proportion of the cases had they been able to cultivate an organism from the blood. Another fact which was brought out was that, although it was known that there need not be a leucocytosis with fungating endocarditis, he was surprised on looking through the reports to find that in the majority of cases the leucocyte count was 4,000, 5,000, or 7,000 per cubic millimetre, and in only about one in ten was there a marked leucocytosis. The cases showed great variability in their duration: one was in the hospital for three months, recovered fair health, went out, came back again later on, and lived over fifteen months from the onset of the acute symptoms. Many cases with a severe pyrexia due to infective endocarditis ran a very long course before they proved fatal; and some cases of simple, acute rheumatic endocarditis, which ultimately cleared up, had pyrexia for weeks. Applying this to the cases which had been brought forward to-night, he was not satisfied that there was sufficient evidence, such as the presence of emboli or of acute nephritis, to be certain that they were cases of infective endocarditis which had been cut short by the use of collargol as suggested. In some the recovery may have been due to the natural course of a non-virulent endocarditis.

Dr. COWAN, in reply, said that the meaning of the terms employed in connexion with endocarditis was somewhat confusing. If one limited the term "infective endocarditis" to cases in which an organism was isolated from the blood, one would limit it to a series of cases which were not at all representative of progressive infective endocarditis. He had no overwhelming belief in the efficacy of collargol in these cases, but in three examples of febrile endocarditis improvement ensued synchronously with its administration, and in two, at any rate, he thought the two facts might be connected.

Therapeutical and Pharmacological Section.

May 3, 1910.

Professor W. E. DIXON in the Chair.

Some Points in the Treatment of Gastric Ulcer.

By CHARLES BOLTON, M.D.

THE treatment of gastric ulcer has, during the past few years, probably given rise to more discussion than that of any other single disease, and many different lines of treatment have been advocated. It is therefore obviously impossible for me in the short time at my disposal to criticize all the different views which have been held. I shall only be able to touch upon a few points in the principles of the treatment in so far as they are affected by the little that we know of the pathology of the disease.

Series of cases treated by different methods are continually being published in order to demonstrate that this or that treatment results in the greatest number of cures. Thus gastric ulcer has been treated by starvation, by immediate feeding, by olive oil, gelatine, and other drugs, with or without external applications, and modifications of each treatment have been adopted by different physicians. In each case the advocate claims that his own method gives the best results. In some cases the average time spent in bed is shorter; in others relapses of bleeding are less common; in others, again, the average loss of weight during the treatment is less than occurs when other methods are employed.

Very few take into account the subsequent condition of the patient with regard to relapses of the symptoms. This discrepancy indicates that collections of cases of this sort are of relatively little value in comparing the efficacy of various modes of treatment. There are several

reasons for this. The diagnosis of gastric ulcer presents great difficulties, and in a large number of cases is impossible. The disease is very variable, both with regard to the condition of the ulcer, its position, extent, and external adhesions, the associated gastric disturbances of motility and secretion, and the condition of the gastric mucous membrane with regard to the presence or absence of gastritis. Acute and chronic ulcer are usually not distinctly separated. The constitution of the patient must also be taken into account. As in health there are individual idiosyncrasies in the matter of diet, so in disease a diet suitable for one patient may be unsuitable for another.

I do not wish to imply that all the different treatments are purely empirical, and that the statistics merely represent attempts to find out what a treatment selected at random is worth, because each treatment is apparently based upon one or more points which the originator considers to be of especial importance in the pathology of gastric ulcer. But I wish to point out that this method of collecting and publishing statistics of cases leads to erroneous results, because different classes of cases which cannot be compared with each other are apparently mixed together. Each case must therefore be studied individually, the pathological conditions present first determined as exactly as possible, and the case then treated according to one or two general principles founded upon our knowledge of the pathology of gastric ulceration, suitable modifications being introduced at the discretion of the medical attendant to meet the requirements of each patient. I am sure that better results will be obtained in this way than if a special "cure" be adopted and applied indiscriminately to every patient as directed.

I will first indicate what conditions the physician is called upon to treat and how such conditions may be recognized. We must first distinguish acute from chronic gastric ulcer. It is very probable that chronic gastric ulcer arises from the acute variety, because occasionally transitional conditions between the two may be found. For instance, acute gastric ulcer may be considerably delayed in its time of healing and it may also gradually extend. The records of such cases are difficult to collect, because the patients do not, as a rule, die, but it cannot be denied that there is a fair amount of evidence in favour of this view. Acute gastric ulcers vary in diameter from about the size of a split pea to 1 in. They come before the notice of the physician either whilst they are in process of formation or when they are healing, and in either case profuse bleeding may occur. In fact an acute ulcer may be almost completely healed whilst bleeding from an open artery in the

centre continues. It may be the original artery which was opened up during the formation of the ulcer and has continued bleeding, or it may have been opened later as the result of digestion of the centre of the base of the ulcer whilst the edges were healing. Acute ulcers are often called superficial erosions because they not uncommonly look quite superficial, although they extend down to the muscular coat. This is not to be wondered at considering that the thickness of the moderately stretched stomach is only about 4 mm. and the mucous membrane 1 mm. They are also often called fissures, because during healing an acute ulcer tends to contract up and form a radiating or a small depressed fissure-like scar. An ulcer in the living stomach may look like a fissure and when slightly stretched assume the usual rounded form.

The tendency of an acute ulcer is to heal rapidly, as in animals, in a few weeks, and also to recur at intervals in the same individual. Such acute ulcers may quite commonly be diagnosed from chronic gastric ulcer, but, as in the case of most other diseases, the diagnosis may be quite impossible.

Acute gastric ulcer is commonly often latent and gives rise to no symptoms. Otherwise it announces its presence in one of three ways: (1) By a sudden perforation; (2) by sudden and profuse hæmorrhage; (3) by pain and vomiting, with or without hæmorrhage. As the ulcers tend to heal quickly, the symptoms last a proportionately short time.

The usual history of a *chronic* gastric ulcer is one of long-standing indigestion, with attacks of pain, vomiting, and hæmatemesis at intervals.

Since acute ulcers tend to recur in the same individual, the recurrences are usually diagnosed as relapses of chronic gastric ulcer, but attention to the following points will settle the diagnosis in many cases. There is no *one* point of distinction, so that the whole group of symptoms must be carefully studied. The pain is not so severe as in chronic ulcer, and it is not so commonly paroxysmal and independent of food, neither is it so liable to be continuous. Vomiting is commoner in the chronic form. Hæmatemesis in the acute disease is more liable to be profuse, to commence suddenly, and to cease suddenly. Hyperacidity occurs frequently in chronic ulcer; in acute ulcer the few estimations which have been made indicate that the acidity is normal or diminished. My own experiments on animals show that when an acute ulcer is in process of formation the acidity is diminished, but when it is healing the latter becomes normal. Wasting is only seen in chronic ulcer. Very long intervals between the attacks are in favour of recurrences of acute ulcer. Diminishing lengths of the intervals, and, finally, a condition

of more or less constant pain and vomiting are characteristic of a chronic ulcer.

Absolute freedom from symptoms during the intervals is in favour of acute ulcer, and the presence of some form of indigestion in favour of chronic ulcer. Of course the symptoms of indigestion may occur from time to time in a normal person, and they often precede the symptoms of an acute ulcer; but this point is certainly of value when taken in conjunction with others. Even though absolutely free intervals occur in chronic ulcer, it will often be found that the patient is unable to eat a full diet, and only keeps well by living on milk, eggs, and fish. In intervals between acute ulcers the patient can more often take any diet with impunity. Acute ulcer is commoner in the young, and especially in women, although no age is exempt. Finally, transitional cases occur, in which the patient may or may not have had a previous attack, but the duration of the symptoms is prolonged. Such cases are instances of acute ulcer, the healing of which is delayed or which is extending. Unhealed ulcers have been found more than two and a half months after the burns which caused them have healed, and many cases are recorded in which acute ulcers following burns have only given rise to symptoms some weeks after the burns have healed. The point I wish to emphasize is that at one end of the scale is acute ulcer which usually heals rapidly; that at the other end is chronic ulcer, and that between the two are ulcers which, beginning acutely, heal slowly, or gradually extend, and some of which become chronic ulcer.

I should like to point out that the healing of acute ulcers may cause considerable deformity of the stomach, and that the character of the scar depends upon the size of the ulcer and the time it takes to heal; so that many scars which have been said to be those of chronic ulcers in the past have probably been those of acute ulcers, or of acute ulcers the healing of which has been delayed. I have gone into these points somewhat at length to show the great importance of acute ulcer, which I believe to be much commoner than is usually taught, and also to emphasize the fact that the more normally and the quicker an acute ulcer heals the less likely are there to be left those sequelæ which necessitate surgical intervention.

TREATMENT OF ACUTE ULCER.

When an acute ulcer *perforates*, the case by common consent should be handed over to the surgeon at the very earliest moment, and success

in the treatment of this condition must largely depend upon recognition of the earliest symptoms of perforation. *Hæmorrhage* from an acute ulcer is, in my opinion, one of the most important conditions the physician is called upon to treat, and it is so common in this disease, and so often the only symptom, that it constitutes a definite type of the malady. It may or may not be possible to diagnose whether the ulcer is in process of formation or is already healing, by the time at which hæmorrhage has occurred during the course of the attack, but I do not think that this is of so much importance since the hæmorrhage has to be primarily treated. Hæmorrhage from an acute ulcer which is at all profuse is always due to perforation of an artery. The sudden cessation of the hæmorrhage is due to plugging of the vessel with a clot. This clot may be interfered with by one of three factors: (1) The condition of distension of the stomach, which opens up and stretches the ulcer; (2) the alternate contraction and relaxation of the muscular coats of the stomach which mechanically dislodge the clot; (3) the gastric juice which digests the clot. Whether the clot be loosened or disintegrated, the blood pressure brings on a recurrence of the bleeding. Sometimes the wall of the artery may project almost beyond the level of the muscularis mucosæ, and then the clot protrudes into the cavity of the stomach.

Such a condition is due to the movements of the stomach and the pressure of the blood in the artery. The aims of treatment in such cases should be (1) to keep the stomach in the contracted state, (2) to prevent movements of the organ, and (3) to keep the digestive glands in the resting condition. In conditions where smaller amounts of blood are lost it must be impossible to say whether the bleeding is from smaller arteries, veins, or merely capillaries, but I think that in all these cases the same treatment should be employed. The patient should be kept at absolute rest in bed in the supine position, and not allowed to move a muscle. Morphia should be subcutaneously administered in all cases of severe hæmorrhage to assist in keeping the patient quiet. The mouth is to be thoroughly cleaned, and pain, if present, treated by morphia, and ice to the epigastrium. For the first twelve hours no feeding is attempted, but after this time has elapsed rectal feeds are started, consisting of peptonized milk and glucose every six hours, the rectum being washed out each morning. When the bleeding has been stopped, for three days mouth feeding is commenced and the rectal feeding discontinued. By stoppage of bleeding I mean not only absence of hæmatemesis, but also of fresh melæna, because as long as fresh melæna is present active bleeding is going on. A simple enema is given every other day

as soon as mouth feeding is commenced, and at the end of a week saline aperients are commenced.

I have never seen a case of parotitis occur if the mouth be kept properly clean.

I do not employ rectal feeding in gastric ulcer in any other condition, because it means that the patient is on a starvation diet; but I think at all costs the stomach must be kept at absolute rest in the case of bleeding from an acute gastric ulcer, and it is not necessary to employ the method longer than for a few days. The time is usually so short that I hardly think it necessary to employ the subcutaneous method of feeding with blood serum or other substances. In this way the stomach is kept in the resting condition both with regard to movement and secretion. It has been stated, and also denied, that rectal feeding causes a flow of gastric juice. I think it is, at all events, likely that the blood in the stomach causes some secretion of gastric juice, and to overcome this difficulty I have allowed the patient to frequently drink lime water by teaspoonfuls. This alkali is preferable to sodium bicarbonate, which distends the stomach by liberation of carbonic-oxygen gas. The alkali neutralizes what acid is in the stomach and does not encourage further secretion. The movements of the stomach which are set up by this fluid must be minimal. In four cases within the last few months at University College Hospital the hæmorrhage of acute ulcers relapsed on commencing mouth feeding and led to the death of the patients. According to Lenhartz [1] when feeding is started by mouth at once recurrence of bleeding only occurs in 6.4 per cent. of patients, but I do not see how it is possible to collect a series of cases in which hæmorrhage is occurring precisely under the same conditions. The giving of food by the mouth neutralizes what acid is present in the stomach, but it excites further secretion, and, as a fact, there is always in contact with the stomach wall a layer of freshly secreted gastric juice which is about to soak into the food. This can be shown by giving a meal soaked in alkali. Although the main body of the food is strongly alkaline, there is always a layer in contact with the stomach wall which is strongly acid in reaction, and this layer gradually deepens until the whole has attained the normal acidity. A digesting stomach is therefore in a condition to disintegrate the clot which is blocking the artery. Muscular movements are also excited by food, and these tend to dislodge the clot.

The icebag of Lenhartz, if it has any influence in diminishing the movements of the stomach, must thereby cause stagnation of the stomach

contents and a more prolonged action of the digestive fluids upon the clot. I believe that when the bleeding is slight, mouth feeding could be commenced at once; but it is impossible to be sure of the size of the vessel, because in a case recently under my care a fair-sized artery was opened, and yet the patient only had coffee-ground vomit: so that I make the rule that, in all cases of hæmorrhage, food should not be administered by mouth. One cannot be altogether guided by statistics, when one sees actual cases in which bleeding breaks out two or three days after the administration of food. On the other hand, I do not state that the patient never dies if fed by the rectum. My own experiments show the importance of the condition of the gastric glands upon the production of gastric ulceration. If the gastric juice be put out of action, ulceration fails to occur. If the stomach is digesting, ulceration produced in it is more marked than when the organ is resting. The presence of food in the stomach must therefore be harmful to a newly-formed clot. It is conceivable that the gelatine treatment of Senator might be of advantage if the bleeding came from the capillaries or small veins, but hardly in the case of serious bleeding from an artery. It does not appear to have found many advocates in England.

The older physicians placed great reliance on the various styptics, and later adrenalin has been used; but I do not think modern physicians use drugs at all largely. I do not myself. In severe cases saline solution, with or without glucose, is, I suppose, administered subcutaneously by most practitioners. The question of operation has been much discussed in relation to profuse hæmatemesis in acute gastric ulcer. A good deal has been written about it, and I think most surgeons are not greatly in favour of it. It may be stated as a fact that the vast majority of cases of hæmorrhage from acute gastric ulcer recover, but that occasionally profuse hæmorrhage proves fatal. I think that fatal hæmorrhage is more common than is usually supposed, judging from the opinions of various writers, and from what I have seen myself.

Whatever is the exact mortality from hæmorrhage does not matter; the fact remains that *sometimes it does* prove fatal. It is not the *first* hæmorrhage which kills the patient as a rule, but *repetitions* due to dislodgement of the clot. I think all will agree that unless the hæmorrhage is so profuse as to seriously threaten life, the case should be treated medically. It is impossible to give exact indications for operation, and each case must be regarded as an individual problem. Such very severe cases recover that it is nearly always impossible to say that a given patient would have died if he had not been operated upon, and many

physicians would not agree with operation in any case. We have three points to go on in dealing with a case: (1) Profuse hæmorrhage is sometimes fatal; (2) in the particular case under consideration the bleeding has reached the danger point; (3) many similar cases are recorded in which the bleeding point has been secured and the patient has survived. The second point is the really difficult one—namely, into what degree of danger is the patient to be allowed to enter before the surgeon be called in.

The question of the mortality of this operation should be put aside in the consideration, as the case is naturally desperate. If a patient vomit 2 or 3 pints of blood, and, after an interval, if the clot break down and a similar amount be vomited, I think the case should be operated upon. But it is quite common for a profuse hæmatemesis to be followed by smaller ones at intervals, or for continued melæna only to follow, so that clear indications may not exist. In doubtful cases the most important point is to be guided by the amount of the initial loss as indicating the size of the ruptured vessel more or less accurately, the condition of the pulse, and the colour of the patient. With regard to the operation itself, there is only one thing to be done—namely, to rapidly open the stomach and secure the bleeding point in the way which commends itself best to the surgeon.

The operation is done as a means of immediately saving life, and gastro-enterostomy is of course useless. The acute ulcer may not be found, as sometimes it is almost healed, or it may be very small and the vessel may not be bleeding at the moment. The surgeon must take his chance of this event. Acute ulcers may be multiple, but it is unlikely that an artery will be opened up in more than one of them, and the most important can at all events be dealt with. The bleeding being stopped, the next stage in the treatment is to put the ulcer under the best conditions for healing. The following remarks apply equally well to the treatment of acute gastric ulcer, whether hæmorrhage has previously occurred or whether the case has merely come up with an attack of pain and vomiting which, from the history and state of the patient, is probably due to an acute gastric ulcer.

In whatever way the initial change is produced which leads to an acute gastric ulcer, we know that the subsequent course of events is greatly affected by the gastric juice. I have been able to show that gastrototoxic ulcers fail to appear at all if the gastric juice be put out of action, and, on the other hand, that hyperacidity of the gastric juice increases the extent of the ulceration, since hydrochloric acid acts as

a protoplasmic poison. A further point of importance is that an ulcer produced in the digesting stomach is very much more extensive than an ulcer produced by similar means in the fasting stomach.

However the ulcer be produced, and whether in man or in the lower animals, the same principles must obtain, since the laws regulating the digestive processes in all animals are the same. It seems that if we wish to have the smallest amount of damage done we must keep the stomach in the resting condition and the acidity as low as possible. Unfortunately, however, we have no clinical means at present of determining whether an acute ulcer in the human being is being produced or is in the condition of healing at the moment when it is brought before us. However, we *do* know that an acute ulcer is rapidly produced, in from twenty-four to seventy-two hours with the gastric juice in its normal condition and the diet normal, so that probably when the case comes under the notice of the physician the ulcer is already fully formed. This probability is strengthened by the results of post-mortem examinations. We further know that the natural tendency of an acute gastric ulcer in both man and the lower animals is to commence healing at once, and for the healing to be complete in a few weeks. This fact is well known experimentally, and is certainly so in the human being, because scars similar in all particulars to those found in experimental animals may be seen in patients who have had short attacks of illness similar to those of gastric ulcer.

Cases are also recorded of injuries inflicted upon the stomach in man which were found to have healed rapidly by post-mortem examination. It is also a very common experience when death occurs from hæmorrhage in acute ulcer to find it advanced in healing, although the illness has been of very short duration. We have seen that occasionally acute ulcers may be delayed in their healing and that sometimes they may extend, so that it is more important from the point of view of the treatment of a case to find out what conditions promote or hinder the *healing* of acute gastric ulcer than what is the actual initial lesion provoking its formation. It is here that experiments upon animals have been of great assistance in affording indications for treatment. It is merely necessary to state here that various experimenters have been credited with proving that hyperacidity of the gastric juice delays the healing of an acute ulcer. In my opinion many of these experiments are of no value, because a definite ulcer was not produced in the first instance, the solutions employed were in some cases too strong, and an insufficient number of experiments were done. My own experiments have shown that so long as the motor

power of the stomach is intact, a hyperacidity of such a degree as is likely to be found in pathological conditions will not delay the healing of an acute ulcer, and I am not alone in maintaining this position, because more recent experimenters have confirmed it.

When the ulcer is being formed, and the gastric juice is acting destructively, hyperacidity will increase this destructive tendency; but when healing has commenced, hyperacidity has no more influence in delaying this than has the normal gastric juice. I have also found that diminished acidity of the stomach contents or moderate infection of the food is likewise without influence. The question is a different one when the motor power of the stomach is disturbed, because when retention of food occurs the healing is definitely delayed for a period of at least twice the normal time. This is due to prolonged contact of the partially digested food with the connective-tissue base of the ulcer, in which necrosis and excessive formation of fibrous tissue is produced, so that the epithelium grows over the base with more difficulty than normally. This being the case, one would expect that an ulcer would not heal so well if the animal were fed on solid food as if it were fed on milk. This is undoubtedly the case, but the effect is seen only in the early stages of healing. When an ulcer is healing, the granulation tissue of the base first becomes covered with a layer of flattened epithelium, consisting of a single layer of cells, and later on this layer thickens and glands develop. Until a continuous layer of cells has formed, the connective tissue of the base is exposed to the action of the digestive fluids and healing may be delayed; but when once the base is covered up, even with a single layer of cells, the remaining stages will go on normally. Meat is retained longer in the stomach than milk, and produces a larger secretion of gastric juice. On a milk diet I have found that the base of the ulcer is covered with epithelium much more quickly than on a meat diet. But I have been surprised to find that diet does not make a greater difference than it does, because even on a meat diet it is only a question of a little delay in the earlier stages. However, there is no doubt that the most favourable diet in the early stages is a fluid one which is easily digested and not retained long in the stomach. The most important principles to be observed are to allow the stomach to empty itself as quickly as possible, and to give a food which produces only a moderate flow of gastric juice. My experiments on animals have convinced me that rectal feeding for any condition except gastric hæmorrhage is to be condemned, and that the milk-and-egg diet of Lenhartz has much to

commend it. In fact, so long as the diet is fluid, to begin with for a week or ten days, and at first given in small quantities at frequent intervals, and so long as solid food is not too suddenly given, I do not think the exact quality of the food matters very much, except that meat extracts should not be used. Lenhartz's method is not altogether founded on correct pathology. His diet is intended to neutralize hyperacidity, which it cannot do, and he puts an ice-bag on the abdomen to diminish the movements of the stomach, which can only lead to delayed emptying of that organ; moreover, I do not think that one definite and fixed diet is suitable for every patient, and it is unnecessary.

I do not think that a better diet than peptonized milk can be used for the first three or four days, in most cases followed by ordinary milk, raw eggs, and chicken jelly, then bread crumbs, pounded fish and chicken, milk puddings, boiled eggs, custard and bread and butter, and, later, minced chicken and mutton, potatoes and sieved vegetables; but I do not wish to be dogmatic in laying down definite rules. I think that the patient should be on a more or less full diet at the end of a month. I have found that beef even after this time is badly borne by most patients, and I think it is best not to allow it for the next few months, during which time I warn the patient to live on the simplest food and properly masticate it, to have the teeth properly attended to, and to rest before and after meals. For the relief of pain I use an epigastric ice-bag when hæmorrhage is occurring, and in all other cases hot poultices.

I have not found bismuth to be of any particular value, but sometimes order dilute hydrocyanic acid, mv in two teaspoonfuls of water, for vomiting. When the patient is convalescent I think some preparation of iron should be given. It is very improbable that any direct connexion between chlorosis and ulcer of the stomach exists, but still the effect of anæmia upon the healing of acute gastric ulcer is an important point, because hæmorrhage leading to anæmia is a common event in ulcer. The fact that an ulcer rapidly heals although bleeding is going on from the centre of it seems to show that the importance of anæmia in preventing healing has been over-estimated. Quinke [7] did two experiments in which lesions were produced in the dog before and after bleeding. In the first experiment the anæmic ulcer took at least thirteen days longer to heal than the normal ulcer. The second experiment proves nothing, because the condition of the ulcer is only described up to the twelfth day. Silbermann's [9] and Litthauer's [3] experiments are not quite comparable to the class of case we are dealing with, because

they produced hæmoglobinæmia by injection of chemicals. The evidence that previous loss of blood delays the healing of an ulcer is, therefore, of no great importance, certainly not sufficiently conclusive to demand the exhibition of iron during the treatment of acute gastric ulcer.

I have so far dealt with the best means of promoting the normal healing of acute gastric ulcer. I have already mentioned that in man acute ulcer may be delayed in its healing, and also that an acute ulcer may gradually increase in extent. Unfortunately, we have no clinical means of diagnosing such conditions at present, except that the history is longer than that of an acute ulcer and hæmatemesis may occur late in the disease. I regard such conditions as intermediate between acute and chronic ulcer, and there is no doubt that one factor in the causation is retention of food, so that these cases should be treated as I have already described for acute ulcer, the time and the graduation of the treatment being somewhat extended.

TREATMENT OF CHRONIC GASTRIC ULCER.

No experimenter has ever yet succeeded in producing a chronic gastric ulcer in an animal, so that we know practically nothing about its mode of extension or the conditions under which it heals. I think that the healing of a chronic ulcer is perhaps less common than is usually supposed, because it is probable that, judging from the extensive scarring which may follow an acute ulcer in animals, many scars which have hitherto been considered to result from the healing of chronic ulcers have really been those of acute ulcers more or less delayed in their healing. We certainly know that, as a rule, the symptoms of a chronic ulcer rapidly disappear with rest and appropriate diet, but how many of these ulcers heal it is impossible to say. Probably not many, judging from post-mortem records and the previous history which usually tells of several previous attacks of a similar nature. In the treatment all we can do is to be guided by the principles we have observed in the treatment of acute ulcer. We cannot, however, expect a thickened chronic ulcer to heal in three or four weeks as an acute ulcer does. It must first be got into such a condition that it will stop spreading and that healing may commence. After this the actual healing must be slower than in the acute form of the disease, so that although the same principles must be adopted, the treatment must extend over a considerably longer period. I was able to show experimentally that in the condition of pyloric stenosis an existing ulcer often took on a spreading character, and rapid

ulceration occurred in the surrounding mucous membrane, and I have lately seen two clinical cases in which a chronic ulcer rapidly spread by acute ulceration of the adjoining mucous membrane in acute pyloric obstruction; sometimes one may see evidences of the same thing in closely examining ordinary chronic ulcers of the stomach. The chief symptom of these cases was coffee-ground vomiting, which apparently denoted acute extension of the ulcer. I think it must be taken for granted that recurrences of hæmorrhage in a chronic ulcer indicate extension of the ulceration, either laterally or in the depth of the ulcer, and there can be no doubt that delayed emptying of the stomach is one cause of such extension.

This is supported by the fact of cessation of hæmorrhage from chronic ulcer after gastro-enterostomy, which, by allowing of the rapid emptying of the stomach, probably stops the extension of the disease. To quote a series of such operations with known after-results, out of twenty-one cases of simple gastro-jejunostomy for recurrent bleeding in chronic ulcer by Moynihan [4], two deaths occurred, and the remaining nineteen patients were free from recurrence of hæmorrhage. A few of these cases were duodenal ulcers; how many is not stated, but the majority were chronic gastric ulcers. Both the cases I have quoted above were operated upon for perforation, and in both cases acute pyloric stenosis was produced, with the result that acute extension of the ulcer occurred. This is a very strong argument in favour of performing gastro-enterostomy in addition to stitching up the perforated ulcer, as advocated by Paterson [5] and other surgeons; although the advisability of this must of course be left to the surgeon, who should decide at each individual operation, according to the conditions found. It is therefore of great importance in the treatment of chronic gastric ulcer as in that of the acute disease to take care that no undue delay occurs in the emptying of the stomach. As in acute gastric ulcer, I think that there is only one condition which necessitates the stomach being kept empty, and in which rectal feeding should be temporarily resorted to, and that is hæmorrhage. The treatment of hæmorrhage from a chronic ulcer I conduct on exactly the same lines as I have described for the acute ulcer.

It is perhaps more necessary to give an alkaline fluid by the mouth in the chronic disease, as a large number of cases have hyperacidity, which increases the destructive action of the gastric juice when an ulcer is extending as it is in recurrent hæmorrhage. In the treatment of a case in which hæmorrhage has ceased, or in which no hæmorrhage has

occurred, we must remember that we have no certain means of telling whether the ulcer is extending, is stationary, or in the condition of healing, so that we must be guided by the two principles that delayed emptying of the stomach increases the tendency to acute extension of the ulcer and delays the healing, and that hyperacidity increases the tendency to ulceration.

Believing, as I do, that the propagation of chronic gastric ulcer is chiefly dependent upon local conditions of the stomach rather than upon the condition of the blood of the patient, I should administer the same diet as in the acute condition, but increase it more slowly and extend the treatment over a longer period, thereby relieving the stomach rather than putting the condition of nutrition of the patient in the first place. It is, of course, impossible for a chronic ulcer to heal in a month, as the acute variety is hardly healed before this time, so that I think the treatment should extend over a period of six weeks, during which the patient is confined to bed, and a further six weeks during which he is allowed to get up. After this he is to eat a light diet for several months, instructions being, of course, given with regard to the care of the teeth, mastication, regular action of the bowels, and exercise.

With regard to the *quality* of the diet, I think that of Lenhartz leaves nothing to be desired. I, however, strongly object to the principle of feeding a bleeding patient, and I do not think solid food should be allowed till the beginning of the third week; milk, eggs, and jellies being the diet for the first fortnight. During the second fortnight the patient should gradually get on to pounded fish, pounded chicken, lightly boiled eggs, thin bread and butter, and custard. During the third fortnight minced chicken, and mutton, potatoes and sieved vegetables, and milk puddings. During the next six weeks he would gradually be put on an ordinary light diet, at which he would remain for the next few months. I give this only as a broad outline, and leave the exact details to be filled in for each individual case by the medical attendant according to his ingenuity.

I think iron ought not to be given as a rule till after the first six weeks, and I do not think that bismuth is of any use. For pain, I think hot poultices are sufficient, except the patient is bleeding, and then an ice-bag should be applied. Vomiting is treated by modifying the diet and by hydrocyanic acid in small amounts of water. The Carlsbad water of von Leube [2] is certainly useful as an aperient, but in no other way. Neither do I think that the constant application of poultices which he so elaborately describes is of any value except occasionally to relieve pain.

In addition he does not keep the patient long enough in bed. The gelatine treatment of Senator [8] does not give any better results in any way than other methods, and it has certainly not become popular. We know of no specific treatment for gastric ulcer, so that any special treatment can be one in name only. As a fact, each of the three special methods gives practically the same results with regard to mortality and immediate results. Statistics are constantly published by many observers, and differ more or less, as would naturally be expected. Von Leube's mortality is variously given at from 0·3 to 4 per cent., Senator's about 4 per cent., and that of Lenhartz from 2 to 3 per cent. Von Leube states that he completely cured 566 patients out of 627, and of these 475 were cured under five weeks. In one group of statistics of patients treated according to Senator twenty-eight out of fifty patients were said to be healed in an average of 20·5 days.

Such statistics as these are obviously of little use, but they show that the special factor underlying each treatment does equally little harm to the patient if it does no good. What we want to know is how many patients are cured as shown by after-results, because cessation of symptoms is no indication that the ulcer has healed. Statistics supplying these facts are scanty. Spriggs [10] gives the after-results of twenty-one cases treated by the Lenhartz method: nine had a definite return of the symptoms of ulcer, and three indigestion. These cases probably included acute as well as chronic ulcer, and it is more than likely that return of symptoms in true chronic ulcer would occur in a larger percentage of cases, whatever the treatment. In other words, I think it is probable that not half the cases of chronic ulcer heal by medical means; according to Paterson's statistics [5], about 26 per cent. are cured. I think a fair treatment, such as I have described above, and extending over three months, should be given; and, if the symptoms return, a surgeon should be called in to perform gastro-enterostomy, which, according to what we know of the pathology of the disease, is a rational procedure and produces its good effects by allowing the stomach to empty itself rapidly.

I am quite aware that symptoms may return after this operation, but the same may be said of gastro-enterostomy for pyloric obstruction, and no one would hesitate in handing such a case over to the surgeon. To sum up, it may be stated that acute gastric ulcer of normal or delayed healing is to be treated by medical means, except those cases which perforate, and perhaps a few in which dangerous hæmorrhage occurs; these latter are to be handed over to the surgeon.

Probably more than half the cases of true chronic ulcer should be treated surgically; but a three months' medical treatment is to be tried first, followed by careful dieting, when, if further symptoms occur (pain, vomiting, hæmatemesis), the ulcer is probably incurable by medical means. All cases of pyloric obstruction, hour-glass stomach, perigastric adhesions and perforation are to be at once treated surgically.

A final word may be added with regard to prophylactic treatment in patients who have recovered from gastric ulcer. We know that bacteria and their products are one important cause of acute ulcer, and it is proved that they do so by attacking the stomach through the blood stream; but there is no evidence to show that they are able to do so from the cavity of the stomach in the ordinary condition of that organ. It is therefore important to remove all possible sources of infection such as pyorrhœa alveolaris, chronic appendicitis, and so on. I need not remind you that in chronic appendicitis, hæmorrhage may occur from the stomach. I have stated above that hyperacidity of the gastric juice is an important predisposing cause of ulceration, and my experiments have demonstrated that other acid substances and protoplasmic poisons, in very weak solutions which are innocuous alone, are able to markedly increase the tendency to ulceration of the stomach. Amongst such acids is acetic acid. I have found that acetic acid of one-eighth the strength of vinegar is able to act in this way, and it is a not uncommon practice amongst a certain class of people to consume large quantities of vinegar. Many other substances which are unconsciously taken in with the food may act in a similar manner.

This side of the subject is quite in its infancy, and these remarks must be rather taken as suggestions for future investigations than as a basis on which to found a definite prophylactic treatment. Still, they show the importance of teaching the patient how to diet himself properly, and to avoid food to which preservatives have been added. It goes without saying that to keep the stomach free from disorders of function must be an important point in prophylactic treatment. In conclusion, it is true that I have told you nothing new in the treatment of gastric ulcer, but I may nevertheless have been able to place the principles of treatment which I have advocated upon firmer foundations than they were before, and if this is so our time to-day has not been wasted.

I thank you for the honour you have done me in asking me to read this paper.

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DISCUSSION.

The CHAIRMAN (Professor Dixon) said all present were greatly indebted to Dr. Bolton for his paper, on a subject which, from the experimental point of view, he had made his own. With regard to the clinical views therein expressed, he (Dr. Dixon) had heard other views expressed before the Society, and probably those would be brought out in the discussion.

Dr. E. I. SPRIGGS said the Section was indebted to Dr. Bolton for giving an account of his researches and of the conclusions he had reached, and not least for the admirable photographs which were shown. He was glad to hear that Dr. Bolton came to no startling conclusions as to treatment, because it was obvious from the different series of cases which had been published on the subject that there was no one method of treating gastric ulcer: the good results seemed to depend largely on the care and thoroughness with which the physician carried out the particular method which he employed. But he dissented from Dr. Bolton's view that published series of cases were of little value. To argue from laboratory work alone was unscientific. He (Dr. Spriggs) was not blind to the importance and value of experimental work. Bridges were erected by the help of mathematical formulæ dealing with strains and stresses, but in the last resort the proof of the soundness of the bridge lay in its capacity to carry the train safely. Similarly in regard to the treatment of gastric ulcer, every proposal must stand the test of experience. It was said that people who collected series of cases might be biased. That was true, but so might the laboratory worker be biased. Except for that protest, he found himself in agreement with most of Dr. Bolton's conclusions. Dr. Bolton was averse to feeding at once in cases of hæmorrhage; he regarded it, like many other people, as an obviously dangerous proceeding which should not be encouraged. The answer was that, so far as experience had gone—and it had extended to hundreds of cases—the frequency of recurrence of bleeding in cases

of gastric ulcer when patients were fed, with discretion, immediately after hæmorrhage was less than where the cases were not fed. The speaker had seen remarkable instances of success. One was a girl, an account of whose case was now in the press, who had recurring hæmorrhage, which was treated on the lines which would commend themselves to Dr. Bolton, including morphia, with the exception that she had once or twice adrenalin by the mouth in white of egg. The bleeding continued, however, and after the sixth hæmorrhage she was too ill for operation. She came under his care, and feeding her with small quantities of protein and fat was tried, with the happiest result. It was known that severe cases of hæmorrhage did sometimes recover, and we could not draw too definite conclusions from individual cases; but that it was not dangerous to give protein and fat after hæmorrhage was supported by statistics and by Dr. Spriggs's experience. Dr. Bolton did not give his opinion of the oil treatment suggested by Cohnheim and by Walko. He (Dr. Spriggs) had used it alone and combined with protein. Since oils and other fats lessened the secretion of juice it was a reasonable treatment. His own conclusion was that the treatment of gastric ulcer by all sorts of methods might be successful; he had tried most of them. It then became a question of the convenience of the method used, and of the comfort of the patient. From this point of view the more we could do without rectal injections the better. He preferred immediate feeding with a fat-and-protein diet in most cases, and was glad that Dr. Bolton had spoken with approval of that plan of treatment.

Dr. HERTZ said he would have thought there was more difficulty in the diagnosis between acute and chronic ulcer than Dr. Bolton had stated. If one went very carefully into the history of cases of perforating gastric ulcer, one could almost always find that there had previously been gastric symptoms. He had inquired carefully in a dozen consecutive cases, and in not one had he failed to get a history of indigestion for varying periods; and at the operation he had not been able to make out any definite relationship between the previous history and the condition of the ulcer which was found. He had been surprised to notice the number of cases in which Dr. Bolton came across death from hæmorrhage from an acute ulcer. He had thought it was so rare for a patient to die from hæmorrhage from an acute ulcer that the question of operation for it ought not to come under discussion at all. His impression was that the cases of gastric ulcer which died, certainly those on which he had made autopsies, were those in which there was a chronic ulcer associated with arterio-sclerosis in elderly people, in whom the sclerosis was such that the arteries could not contract. In patients past middle age with hard arteries, in whom there was hæmorrhage, he was inclined to advise operation. He agreed with Dr. Spriggs's remarks as to feeding after hæmorrhage. When severe hæmorrhage took place much blood was present in the stomach, and this would excite the secretion of gastric juice. The addition of food, and especially of oil, which inhibited the flow of gastric juice without exciting much movement, could only do good, and he agreed with Dr. Spriggs that statistics

showed that feeding the patient immediately after a hæmorrhage was in fact quite devoid of danger. It was impossible to believe, as Dr. Bolton suggested, that hyperacidity did not prevent healing. Thus every form of treatment which had been found of use had as its basis the keeping of the free hydrochloric acid as low as possible. Not only did healing then occur more rapidly, but there was less pain when the free hydrochloric acid was brought to a minimum. With regard to trying to neutralize the hydrochloric acid by alkalis, it was certainly true that free hydrochloric acid was in contact with the mucous membrane, but it had been proved that the juice secreted by the pyloric end of the stomach was alkaline, and it was at the pyloric end that ulceration chiefly arose, ulcers at the fundus of the stomach being comparatively uncommon. However large a chronic ulcer might be, it was possible for it to heal, and he disagreed with what Dr. Bolton said as to the likelihood and the frequency of healing. There had been cases in which, at operation, a large chronic ulcer had been found, and when a subsequent operation had to be done all trace of the ulcer had disappeared, and only a small scar was visible. It was a common experience that there were intervals, perhaps of months, especially in the summer, when no trace of pain was present, so that it was highly probable that during these periods the ulcer was healed. In treatment one had to deal with the tendency to ulceration, and the particular method of treatment adopted during an "attack" was of comparatively small importance. It was important to give the patient instructions to prevent recurrences by keeping the free hydrochloric acid in the stomach as low as possible, and avoiding all sources of mechanical and chemical irritation of the gastric mucous membrane.

Dr. LANGDON BROWN said he agreed with the former speakers in two particulars: first, in thanking Dr. Bolton for his admirable paper; and, secondly, in saying that, though it might be dangerous, as Dr. Bolton asserted, to indulge in immediate feeding in gastric ulcer after hæmorrhage, he had done it, and others had done it in hundreds of cases, and hæmorrhage did not recur then any more frequently than under rectal feeding, which was equivalent to starvation. He had treated twenty cases on the Lenhartz method, and in only one was there a slight recurrent hæmorrhage; and hæmorrhage had recurred in that case when treatment had been carried out on orthodox lines. If one compared the nitrogen output on rectal feeding, it was found to be no better than if only salines were given *per rectum*. The acidosis might become severe, and it was not easy to get the acidosis abolished by giving dextrose *per rectum*. Really, in rectal feeding one was starving a patient who was already depleted.

Dr. HERBERT FRENCH said there was one small point which he would have liked to have heard more insisted upon—namely, the treatment of the anæmia which was so common in cases of gastric ulcer in women. Dr. Bolton said that anæmia might have some relationship with the occurrence of ulceration, and he drew indefinite conclusions from the experimental results

that had been obtained. But clinically it seemed that anæmia predisposed to ulceration, or at least delayed the cure of an ulcer; if one could make the patients more healthy and less anæmic, that assisted the cure of the ulcer and tended to prevent its recurrence. The difficulty in connexion with the treatment by iron was the tendency of iron compounds to upset the stomach, so that many patients with gastric ulceration appeared to be able to take iron less well than could normal people. One form of iron which seemed to have that drawback to a minimum extent was the alginoid form, which could be given as a powder; it could be given in 10 gr.-doses without upsetting the gastric functions, even when its exhibition was begun within a day or two of an acute hæmatemesis.

Dr. BOLTON, in reply, said that most of the points taken up by the speakers were small ones; and he had not gone in for details, but treated the subject on broad lines. Dr. Spriggs criticized the way in which he had spoken of statistics, and he quite expected he would do so. But Dr. Spriggs merely stated his case, and did not give proof that Dr. Bolton was wrong or that he himself was right. Of course it was of very great value to have series of cases published, and it was the only way to find out what treatment was accomplishing. But the investigator should say he had treated so many *acute* cases and so many *chronic* ones by different methods; that he had treated so many cases in which a *large* hæmorrhage occurred, and so many cases in which there was only capillary *oozing*, and the results were such and such. But what they did was merely to say that they had treated so many cases of gastric ulcer. He (Dr. Bolton) had said that he did not believe that a case of bleeding would be harmed by immediate feeding unless it was due to a large artery. But one never knew whether it was a large artery or not. He had depicted on the screen a case in which a large artery was eroded, but there was only present coffee-ground vomit. That uncertainty was a very important point. Often it was impossible to diagnose whether an ulcer was acute or chronic. He did not believe that the gastrostaxis of Hale White had been proved to exist; he believed that most cases so-called were acute ulceration, an ulceration which was very liable to be missed. There might be only a little scar with a bleeding point in the middle, and if it was not looked for by special methods it would not be found, and the case would be regarded as one of gastrostaxis. Dr. Hale White quoted 29 cases, in 10 of which it was said that the stomach was found on post-mortem examination to be free from ulceration; 19 of these were of no value because the stomach was examined at operation, and it was not always possible for the surgeon at operation to know what was an ulcer and what was not. On stretching out a stomach in which there were multiple bleeding points it would be found that there were ulcers. He therefore regarded gastrostaxis clinically as acute gastric ulcer. The object of his paper was to stimulate the scientific method of employing treatment. First, one had to diagnose the condition as nearly as possible, and then treat it accordingly; if one published 200 or 300

cases of acute ulcer treated by the Lenhartz method, the results would be of the greatest value; but merely saying that so many cases of ulcer had been treated, without regard to whether they were acute or chronic, was not of much value. [Dr. SPRIGGS pointed out that in his series the clinical history was given in every case; the Press did not publish those, but they appeared in the *Proceedings of the Royal Society of Medicine*.¹] Dr. Bolton stated that he would examine the records, but that Dr. Spriggs in his conclusions had grouped all the cases into one class. With regard to hyperacidity and its effect on healing, in the ordinary human being there were other things present besides hyperacidity—such things as motor insufficiency, and so on. It was not merely a question of the hydrochloric acid, though that was of the greatest importance when an ulcer was extending, but one did not know whether it was extending or not. He agreed that a large number of chronic ulcers must heal, but the vast majority of such cases did not heal unless there was some operative procedure. At the end of his paper he dealt with prophylaxis, but there was not time to read it. In answer to Dr. Langdon Brown, he could only repeat that he did not think feeding after hæmorrhage did any harm in many cases; but where there was an artery bleeding, as in the condition he showed on the screen, it must be best to put the patient on rectal feeding for a few days; there was not time in those days for much acidosis or any other intoxication to occur, and he had not seen any ill effect. He was in the habit of treating many gastric ulcers, but, for the reason he had given, he did not attach much value to statistics until they were based upon a much more accurate diagnosis. With regard to chlorosis, one saw ulceration occurring, both acute and chronic, in young and old people, in men and women, and in the absence of chlorosis, and that fact seemed to show that chlorosis was only a concomitant circumstance. He agreed that everything should be done to feed up the patient, and he thought the Lenhartz diet was the best, but he disagreed in thinking that cases should not be treated in that way when there was a large artery bleeding and one was not sure of what was going on. With regard to operation, it seemed natural, in a case like that shown on the screen, to suppose that the only thing to do was to tie the bleeding point. Some surgeons were in favour of operating, while others were not. One had, however, usually no right to say that a patient had been cured by operation because he recovered from the bleeding.

¹ *Proc. Roy. Soc. Med.*, 1909, ii (Therap. and Pharm. Sect.), pp. 94-107.

A Criticism of some Principles in the Treatment of Chronic Nephritis.

By W. LANGDON BROWN, M.D.

I SUPPOSE it would be generally agreed that the orthodox, conventional treatment of chronic nephritis includes the following principles:—

(I) Severe restriction of protein intake, with absolute exclusion of foods rich in albumin, such as eggs. In severe cases, absolute restriction to simple milk diet.

(II) The estimation of the amount of urea in the urine is taken as a guide to the capacity of the kidney.

(III) The kidney is stimulated to increased excretion by the use of diuretics.

(IV) Elimination by the skin is promoted by the use of various diaphoretic measures.

I make so bold as to assert that each of these principles contains, and indeed is based upon, a fundamental fallacy.

In Bright's disease we have probably tended to lay too much stress on the albuminuria. In the chronic parenchymatous form no doubt the drain on the albuminous constituents may become serious, and a secondary anæmia results. But such high degrees of albuminuria are uncommon, and I think we are beginning to realize that this one symptom has unduly dominated our conception of the disease. Von Noorden thinks that any wasting is just as much explained by the monotonous diet as by the loss of albumin.

(I) SEVERE RESTRICTION OF PROTEIN INTAKE.

When we proceed to limit the protein diet rigidly in Bright's disease, are we not led away by false analogies with glycosuria? Whereas there are the following essential differences:—

(1) The sugar can be replaced by other things in a diet, while the protein cannot.

(2) The sugar excretion is preceded by an excess of sugar in the blood; albuminuria is not preceded by excess of albumin in the blood. The latter is due to a kidney lesion, the former is not.

(3)* Recent work shows that there is a great breaking-down of the protein molecule into its constituent groups before it is absorbed into the body. The simple conception of Liebig, according to which the protein food is simply hydrolysed into peptone, and then assimilated into the tissues with no further change than dehydration, is no longer held.

It has been well said that, just as a Gothic cathedral could not be built out of a classical temple without reducing it to its constituent stones, so the protein of the tissues cannot be built out of the protein of the food without splitting it up into its simple constituent groups.

The evidence that albumin (as, for instance, egg albumin) is ordinarily absorbed as such, and is able to "run through" the body, will not really stand investigation. Certain experiments by D'Arcy Power upon himself are usually quoted as evidence of its occurrence. It is true that albumin appeared in his urine on the first day of the experiment, after twelve eggs had been eaten, but it disappeared in the evening, and did not reappear till the afternoon of the third day, after the consumption of forty-eight eggs. It was found again during the evening of this day, after which it disappeared again, and had not reappeared at the end of the experiment, by which time sixty-five eggs had been taken. Only once was the albumin present in sufficient amount to enable its coagulation-point to be determined. It will be noted that there is very little connexion between the number of eggs consumed and the amount of albumin. A very significant point, which seems to have escaped notice, is that at this time D'Arcy Power was aged 22, and that the occurrence of the albuminuria was always observed after a considerable amount of exercise had been taken. Now, transient albuminuria after severe exercise is common in young men, without excess of albuminous diet.

It is difficult to believe on theoretical grounds, therefore, that the albuminuria of nephritis can be influenced by the amount of albumin in the diet, and I have tested the point practically in a good number of cases of chronic nephritis. The following is an instance from which it will be seen that increasing the amount of egg-albumin in the diet did not increase the albuminuria. These figures represent the average of several days on the same diet, so as to avoid the disturbance which would be introduced by the diet of one day not being eliminated till the next.

Diet		Albumin nitrogen	Total nitrogen, less albumin nitrogen
Milk diet and one pint of beef-tea	...	0.562 grm.	6.17 grm.
" " only	...	0.6	7.46 "
" " and one egg	...	0.69	9.77 "
" " and two eggs	...	0.7	7.91 "
" " and three eggs	...	0.649	7.6 "

(II) UREA EXCRETION AS A GUIDE TO THE CAPACITY OF THE KIDNEY.

If the amount of urea excreted be a guide to the capacity of the kidney, the ratio $\frac{\text{urea nitrogen}}{\text{albumin nitrogen}}$ ought to rise if the patient be getting better, and fall if he be getting worse. But I found that this was by no means the case. For consider what happens when we give a normal individual an excess of protein food: he turns it into urea, and excretes it as quickly as possible. The mere fact that the nephritic can turn his protein into urea does not prove he has done any good with that protein. Indeed, evidence is accumulating that such protein excess never gets built up into protoplasm at all.

For this reason it seems to me that the amount of urea excreted in the day gives very little information as to the severity of a case of Bright's disease unless the diet is carefully taken into consideration.

The procedure often adopted is irrational. A man with chronic nephritis on a restricted diet has his urea estimated. Instead of the normal 30 grm., he is found to be passing, say, only 16 grm. The physician concludes that the capacity for urea excretion must be seriously decreased; the patient must take less nitrogenous food. This is done, and the next analysis shows an even lower urea excretion. "Worse and worse," thinks the physician; "this man is only fit for a milk diet." Accordingly he is given 3 pints of milk a day and nothing else. He now bids fair to fulfil the gloomy prognosis formed, unless he fortunately rebels against this pitiful fare, and takes the law into his own hands. For, as the greater part of the urea comes direct from the food, the more the nitrogenous food is restricted the less urea will be excreted.

Of course, a patient on the diet ordinarily given in nephritis passes less urea than normal, because he is given a diet poor in protein. But the output probably will not be so little as that of a healthy fasting man, while it will certainly be more than that of a man on Folin's diet of starch and cream, in which the nitrogenous excretion is reduced to a minimum, because so much of the energy is derived from sources other than protein.

What the physician expects to learn from the urea estimation without reference to the amount of nitrogen in the food is hard to say. If he knew the total nitrogen excreted, as estimated by Kjeldahl's method, he could see whether the body was converting a due proportion of the nitrogen into urea; this would give him some information as to the

capacity of the individual, but urea estimations by themselves tell him practically nothing. This fallacy vitiates many of the conclusions arrived at by so careful an observer as the late Professor Foxwell in his address on "The Estimation of Urea."¹ He hardly mentions the diet factor at all. In one case he certainly says that he found a patient with chronic nephritis passing as much as 585 gr. (39 grm.) of urea a day. On inquiry, he learned that the patient, feeling run down, was taking six meals a day, three of them being good meat meals. This shows that a chronic nephritic can excrete even more than a healthy man does on his ordinary diet, though it presumably taxes his kidneys more. When Professor Foxwell said that a daily output of 250 gr. of urea is the lowest on which a man can permanently exist without losing ground, we can agree with him; for this would represent a daily intake of about 50 grm. of protein, which is little enough to satisfy even the most extreme "nitrogen economist." In short, the amount of urea excreted by the kidney depends on the amount of protein eaten, and within wide limits, on little else.

We know now the physiological minimum of protein is much less than the 100 grm. to 125 grm. formerly ordained. Chittenden's experiments on the effect of a reduced protein diet are by now familiar to all. He has certainly proved a point of great interest and importance—that the minimum protein requirements of the body are much less than was supposed. But he goes much further, and maintains that the minimum is also the optimum. To consume protein in excess of that required for the repair of the tissues he regards as a physiological sin, the wages of which is migraine in earlier life and cardio-vascular degeneration in later. He gives no evidence of this, but assumes that the nitrogenous excess overtaxes the kidneys by which it has to be excreted. It is hard to see why he assumes that the kidneys are unable to do more than the minimum necessary without damage to themselves. He might as well assert that the deeper breathing necessitated by reasonable exercise dangerously overtaxes the capacity of the lung to excrete CO_2 , and tends to asphyxia. He is, in fact, obsessed with the old idea that the body is unable to make any other use of protein food than to repair tissue waste—an idea which other lines of work have rendered improbable. The physiological minimum is not necessarily the physiological optimum. Experience goes to show that where there live, side by side, a race living on a protein-rich diet and one on a protein-poor diet, such as Europeans and natives in India, the morbidity and mortality of an epidemic are

¹ *Lancet* 1908, ii, p. 1425.

much higher in the latter. The rapid rise of Japan corresponds to the adoption of a more liberal nitrogenous diet. To this Chittenden answers that prosperity causes an individual or a race to elaborate the menu, that the increased food is not the cause of the improvement. But undoubtedly the revision of the dietary was responsible for the practical stamping out of beri-beri in the Japanese navy, which formerly diminished its effective strength by at least 25 per cent.

It is curious also to find it stated that all Chittenden's disciples have turned apostates, and have resumed the ordinary diet.

We may conclude that of our protein diet very little is used for direct repair of tissue waste, but doubtless much of the rest is used as a source of energy, and it is at least probable that the ammonia groups set free from this protein excess are useful in neutralizing acids that might otherwise lead to acid intoxication.

What is the bearing of all this on the dietetic treatment of chronic nephritis?

Too rigid a limitation of the protein diet with the idea of diminishing the albuminuria is bad, because it cannot effect the desired object, and deprives the patient of an essential form of nourishment. On the other hand, an excessive protein diet is inadvisable, even if the patient can metabolize it, because he is getting the energy in a form that throws work on to the damaged excretory organs. What is the happy mean? I would suggest that we can arrive at it theoretically in the following way:—

Chittenden's diet gives us the physiological minimum of protein. As the amount of protein in the diet has no appreciable effect on the amount of albumin in the urine, a patient with nephritis would not be able to maintain his nitrogenous equilibrium on Chittenden's diet. We must add to this diet an amount of protein equal to the albumin lost in the urine,¹ when we shall be giving just enough to maintain equilibrium and yet not be taxing the kidney by calling upon it for any unnecessary work.

Von Noorden finds, clinically, that the chronic nephritic can easily excrete up to 15 gm. of nitrogen in the day, but above this elimination becomes irregular and uncertain. Fifteen grammes of nitrogen correspond to 94 gm. of protein, and this is the maximum that should be allowed, while the minimum is about 60 plus the amount of albumin

¹ A convenient rule is this: When the reading of the albuminometer is 5 and the amount of urine is 2 pints, the patient is excreting as much protein as is contained in one egg. I take these figures because they admit of simple proportional calculation, and also because they represent the amount of albumin excreted in a case of chronic parenchymatous nephritis of average severity—i.e., 6 gm., which is the amount of protein in one egg.

in the urine. Thus the theoretical and practical results agree fairly closely.

We should naturally avoid meat extracts and cellular organs, such as sweetbread, because they contain a large proportion of purins which, though useless for nutrition, have to be excreted by the kidney, and, according to von Noorden, the damaged kidney excretes uric acid with difficulty. This is contrary to the principle of physiological rest. But we must equally avoid the monotony of diet which leads to failure of appetite and consequent wasting, while it is incapable of affecting the albuminuria. We can safely permit a much greater variety of diet than is allowed on the orthodox lines. For instance, I believe from my analyses that eggs and things made with eggs certainly may be allowed.

It is undesirable to restrict such patients to milk, which is too dilute a form of food for them, and may increase the œdema. Salt should not be allowed, since it is badly eliminated in many cases of nephritis, and, accumulating in the tissues, increases the œdema by raising the osmotic pressure. Indeed, as Bryant found, even a man with healthy heart and kidneys may develop œdema as the result of taking excess of salt. The substitution of butter and lemon-juice will usually satisfy the patient.

In following this plan we shall avoid adding to the miseries of sufferers from an incurable disease by enforcing unnecessary restrictions. If it be desired to guard against the dangers of possible nitrogen retention, Ernberg's plan may be followed of interposing periods of a week or a fortnight during which a diet poor in protein is taken. But prolonged nitrogen starvation is as bad for a nephritic as for anyone else.

The rules which guide us in acute nephritis or in exacerbations of chronic nephritis are somewhat different, however. "In acute affections we concentrate our attention on the diseased organ, whilst in chronic cases we keep the general condition of the patient more in view" (Von Noorden). Nitrogen retention is a very prominent feature of acute nephritis, and a diet poor in nitrogen is strongly indicated. This period of retention is usually short; if it continues, it is very ominous. A few days' comparative nitrogen starvation will do no harm, and may avoid grave danger.

Von Noorden is of opinion that in acute and dangerous cases this is very necessary, and gives nothing but sugar, water, and fruit-juice for from three to eight days.

It may be noted here again that the degree of albuminuria gives no real clue as to the gravity of the condition. At my suggestion,

Dr. F. W. W. Griffin examined the nitrogenous excretion in a series of cases of scarlatinal nephritis from the beginning. He found that, whereas there was a general relation between the amounts of water, urea, and total nitrogen excreted, there was none between these and the amount of albumin excreted. He concluded that the albumin afforded no more than a danger-signal at the commencement of the condition, and could not be accepted as a trustworthy indicator of the excretory capacity of the kidney.

(III) THE STIMULATION OF THE KIDNEY TO INCREASED EXCRETION BY DIURETICS.

There has always been a tendency to regard flushing out the kidney as a good line of treatment in Bright's disease; but before employing it we should consider what method of diuresis we mean to employ, how far such methods are desirable in the case before us, and how far they will achieve the end desired. Routine and indiscriminate "flushing out" is to be condemned.

Methods of producing Diuresis.

The following are possible:—

(a) By vaso-dilatation in the kidney, as by the caffeine group of drugs. These probably act as direct stimulants to the renal epithelium, the vascular change being secondary.

(b) By vaso-constriction elsewhere, in consequence of which the blood-pressure is raised and more blood is forced through the kidney—e.g., digitalis.

(c) Increase in quantity of circulating fluid—(i) by absorption of water from the intestine, as by giving the patient large quantities of fluid to drink; (ii) by increasing the osmotic pressure of the blood. The saline diuretics, citrates, acetates, &c., act in this way, attracting water from the tissues into the blood-stream.

How far are these Methods desirable in Nephritis?

(a) Why stimulate a damaged structure? I believe I have seen caffeine, theobromine, and diuretin all produce bad effects. It is chiefly in chronic parenchymatous nephritis that one sees them employed, and there is a danger that they will cause a return of acute symptoms; hæmaturia not infrequently follows. I have gradually come to the

conclusion that this group of drugs is unsuitable for nephritis, and should be restricted to cases where diuresis is required and the kidneys are not organically diseased.

(b) *Digitalis* : as the blood-pressure is already raised, why raise it any further ? I recently saw a case of chronic interstitial nephritis with dilating heart, which was causing a diminished urinary secretion. Digitalin injections were being given. I took the blood-pressure, and found it was 200 mm. The patient's whole difficulty was that the heart could no longer work against such high blood-pressure. The effect of the treatment was to load an overworked heart still more. I suggested nitro-glycerine and strophanthus instead ; the pressure fell, and the patient was relieved for the time. I believe the unsatisfactory results which some say *digitalis* gives them are due to its being employed in unsuitable cases such as this. It is, indeed, difficult to see how *digitalis* could be a satisfactory diuretic in cases of nephritis.

(c) In acute nephritis it is really no good to give large quantities of water with the idea of flushing the kidney, for the kidney cannot excrete it, so that it accumulates in the tissues, increasing the œdema.

This defective adjustment of the kidneys to varying water supply is an important clinical point, as the following example, quoted by von Noorden, shows : A normal individual, with an average hourly diuresis of 52 c.c. excreted an average of 723 c.c. for three hours after drinking 1,800 c.c. of *Salvator* water ; under the same conditions a patient with acute nephritis, excreting 91 c.c. hourly before, only passed 103 c.c. after. Spontaneous diuresis is the first and surest sign of convalescence.

The attempt to increase the urinary flow by increasing the osmosis into the blood is less open to objection in acute nephritis. Citrate of potassium renders the urine less acid, and therefore less irritating to the kidney. As the extra water is drawn from the tissues it will tend to diminish, and cannot increase, the œdema. I would put it this way :—

(a) In acute nephritis we cannot flush out the kidney, because the inflamed organ will not respond. I believe that potassium citrate is the best drug, because it does not irritate the kidney, and any diuretic effect it may have is at the expense of the œdema.

(b) In chronic parenchymatous nephritis the kidney is more responsive, but it is undesirable to increase its secretion, either by irritating it by caffeine and the like, or by increasing the already raised pressure. Yet diuresis is here certainly desirable, because, as I have stated, the total excretion of urea follows pretty closely the excretion of water.

Here, again, potassium citrate seems to be freest from objection. Another useful combination is liquor ferri acetatis and liquor ammonii acetatis.

(c) *In chronic interstitial nephritis* the kidney responds quickly to altered intake of water. But some years ago von Noorden claimed that, rather than trying to flush out the kidney, it was desirable to restrict the fluids to $1\frac{1}{2}$ litres a day. He maintained that this did not diminish the urea excretion, while the work of the heart was spared. He considered that the polyuria was secondary to polydipsia. I have tried this plan in a good many cases since, and am inclined to agree that moderate restriction of fluid has advantages over the flushing-out method.

(IV) ELIMINATION BY THE SKIN.

I do not feel in a position to assert so dogmatically that this is always a mistake. In cases of uræmia it may be imperative to promote diaphoresis. But it is open to the following objections:—

(a) Very little nitrogen can be got rid of through the skin compared with the amount that can be eliminated by the bowel.

(b) Physiological rest for the kidney is not secured by giving it a highly-concentrated urine to deal with, for, as already urged, defective adjustment of the kidney to varying concentrations is a prominent feature of nephritis.

(c) Diaphoresis is an exhausting process and depressing to the heart. This is particularly the case with pilocarpin. Although I have occasionally seen good results from its use, I have also seen much harm done, and I have reluctantly come to the conclusion that it is much too risky a drug to employ.

DISCUSSION.

Dr. J. GRAY DUNCANSON said the Section was much indebted to Dr. Langdon Brown for his able address. It was good that the Section should interest itself in the subject of diet as well as in the actions of drugs, and it was evident from what had been said that day that dietaries as rigid as the laws of the Medes and Persians were to be a thing of the past.

Dr. H. CHARLES CAMERON said the Section was much indebted to Dr. Brown because, when unavoidable circumstances had prevented his giving the paper promised, rather than disappoint the Section he had come forward with another. He would have thought that the last two methods of treatment to which Dr. Langdon Brown referred were more directed against the common

symptom of nephritis—œdema—than against the nephritis itself. He had recently been watching closely the results of giving patients widely different amounts of fluids, and his conclusion was that each case had to be settled on its merits. There were cases, in other respects showing a high degree of nephritis, which still retained the power of passing out fluid. He would have thought that from diaphoresis one would not expect benefit so much in the excretion of nitrogen as in the diminution of the œdema. And he suggested another reason which, theoretically, would militate against advising active diaphoresis. Before the fluid could be excreted by the skin the toxins contained in the dropsical collection had to be reabsorbed into the circulation. He certainly thought he had seen cases where excessive diaphoresis was bad for the patient.

Dr. E. I. SPRIGGS said he had found the hot-air bath a valuable remedy for people suffering from nephritis with threatening uræmia. He knew nothing which would, on occasion, produce such an effect in relieving headache and other subjective symptoms. He did not know whether Dr. Langdon Brown's remarks on diaphoresis were intended to discourage the use of hot air. With that possible exception he was in agreement with his interesting paper. In the Oliver Sharpey Lectures of 1906 he had come to the same conclusions, probably largely on the same evidence. He did not think, with Dr. Duncanson, that the days of strict diet were gone. It was essential to know about how much protein was being given to patients with kidney disease. The amount of urea in the urine was a guide to the amount of protein which was being taken, rather than to the functional capacity of the kidney. It was necessary, as long as life lasted, that the body should use and the kidney pass out enough nitrogen for the maintenance of activity; but it was undesirable to give more than was actually necessary. The 60 gm. to 90 gm. of protein mentioned by Dr. Langdon Brown were sufficient, and it required a careful adjustment of diet to know that enough, but not too much, was being given. With regard to eggs, it had been shown that albumin was excreted as such sometimes in the urine, by the precipitin test, but only when albumin had been taken in such large quantities that it was absorbed direct into the blood. That had been supported by injecting albumin into the blood. If injected rapidly it appeared in the urine, but if injected gradually it did not. The conclusion was that if eggs were mixed with ordinary foods they would not increase albuminuria. He would like to support what Dr. Brown had said about red and white meat. With regard to the question of albuminuria in prognosis, interesting observations had been made by Emerson, in America, and published in the *Johns Hopkins Reports*.¹ He found that the best guide was an alteration in the percentage of albumin. The case was different from that of sugars, in regard to which the daily quantity was important. He did not give diuretics in nephritis, except sometimes potassium citrate. Mention had been made of the view that the thickening of the vessels in nephritis was

¹ *Johns Hopkins Hosp. Rep.*, Balt., 1902, x, p. 323.

due to their having to deal with such large quantities of fluid owing to patients drinking a great deal. But that view was untenable. In diabetes there was much greater thirst, and more fluid was being dealt with by the patient day after day; and although there was arterial sclerosis in diabetes, yet such hypertrophy of the heart and of the vessels as in chronic nephritis was almost universally absent. It was easier for the kidney to excrete a dilute fluid, as Dr. Bainbridge had pointed out: hence in granular kidney fluid should not be restricted, but the patient allowed to have what he required, within reason, so long as the quantity of urine was being excreted.

Dr. R. PARAMORE, speaking as a general practitioner, said the medical man was sometimes in a difficulty as to the teaching on the subject now being discussed. The Turkish bath had apparently done good in kidney disease, but it would appear that the urine was more concentrated after it, so that the kidneys might be more irritated by the action of hot air on the skin. He had used pilocarpine in nephritis with apparent benefit; there had been profuse perspiration following. He was not sure whether passing distilled water through the kidneys and giving them rest always produced the expected benefit. He had kept patients on apples and water for days, but was not impressed by the result. He was sure some cases of apparent nephritis had got well on simple oatmeal and milk, with plenty of fruit and vegetables, this treatment being based on the belief that both alcohol and meat were bad for the condition. In some quarters there was a tendency to say whatever had been wrong; old practitioners gave mercury for many things, and, though they did not know its exact action, there were good results. They even gave it for pneumonia. Some said digitalis did harm in kidney cases, others that it was harmless. But experience went a long way after all. Everything could not be tested in the laboratory. In hæmatemesis he gave opium by the mouth and rectum, and rested the stomach and bowel, and later he gave iron, in the shape of the acetate or ammonio-citrate. Many people decried empiricism, but he thought it would be practised for a long time yet.

Dr. LANGDON BROWN, in reply, said that he favoured moderate limitation of fluids in granular kidney, but he did not agree with cutting them down to a smaller quantity than 1,500 c.c., and he did not regard the hypertrophy of the heart as secondary to the amount of fluid consumed. The whole point was that in nephritis the kidney had a defective power of adjustment of the amount of water taken in. The ordinary healthy person would concentrate his urine by a Turkish bath, and his kidney could adjust itself to the varying quantity of water taken; whereas the nephritic's power of doing so was very limited.

INDEX

PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE, Vol. III, 1910

INDEX

(Prepared by Mr. A. L. CLARKE)

Note.—The references are to the Sectional *Proceedings*, which are paged independently. The *Proceedings* of the Society as a whole are indicated by the letters R.S.M., and are placed first in the bound volumes under the heading "General Reports." They are followed by the *Proceedings* of the Sections, which are arranged alphabetically.

- Aachen, treatment of syphilis at, Surg. 216, 243, 245
AARONS, S. JERVOIS.—Specimen of ectopic gestation, Obst. 130
Abdomen, operations on, choice of anæsthetic for, Anæsth. 72
—, pendulous, with atony of bladder, Electr. 147
—, pericarditis due to pneumococcal infection from, Med. 81
—, tumour in, disappearing, case of (P. Sargent), Clin. 85
ABERCROMBIE, P. H., and MCKENZIE, D.—Hysterical deafness with active vestibular reactions, Otol. 74; discussion (p. 75): Dr. Law, Mr. Scott, Dr. McKenzie (reply)
Abortifacient, phosphorus taken as, fatal case of poisoning with unusual subcutaneous hæmorrhages (R. G. Hann and R. A. Veale), Therap. 64
ABRAHAM, PHINEAS S.—Case of alopecia areata (exhibited by J. E. R. McDonagh), Derm. 55
—, case of dermatitis herpetiformis, Derm. 24
—, — of mycosis fungoides, Derm. 39; discussion (p. 40): Dr. Pringle, Mr. McDonagh
—, — of probable pemphigus vegetans, Derm. 23
—, lichen planus in a child (exhibited by J. E. R. McDonagh), Derm. 89
Abscess, alveolar, acute, treatment by vaccine, R.S.M. 87
— following calomel injection in syphilis, Surg. 213
— of ovary, probably due to infection by the *Amæba coli* (H. Williamson and J. D. Barris), Obst. 152
Abscesses, case of, tuberculous nature diagnosed, R.S.M. 95
Accessory sinuses (nasal), suppuration, treatment by vaccine inoculations, R.S.M. 52, 53
Accouchement forcé, Obst. 174
Acetanilid, Therap. 88
Acetic acid, in weak solutions, ingestion of, factor in production of gastric ulceration, Therap. 132
Acetone, presence in breath and urine in recurrent or cyclic vomiting, Child. 79, 82, 87
— — — in starvation, Child. 82
Acetyl salicylic acid, "aspirin" fancy name for, Therap. 100
— — —, confusing list of synonyms for, Therap. 87
Acholuric jaundice, case of (F. H. Jacob), Clin. 157
Achondroplasia, two cases of (R. Milne), Clin. 54
— in twin (R. Hutchison), Child. 41
Acid, resistant powers of teeth to action of, Odont. 75-77.
Acidosis, relation of acute starvation to, Child. 82

- Acids causing dental caries, enamel sole barrier against, *Odont.* 73
 — of decay, power of resistance of enamel to, *Odont.* 78, 81, 83
- ACKERLEY, R.—Discussion on arthritis deformans, *Baln.* 98
 —, observations on the condition of the mouth in 1,000 cases of chronic disease, *Odont.* 94-103; discussion (p. 104): Mr. W. Hern, Dr. H. Macnaughton-Jones, Mr. C. E. Wallis, Mr. Hunt, Mr. J. G. Turner, Dr. W. J. Midelton, Dr. J. Galloway, Mr. P. Daniel, Mr. Sturridge, Mr. L. Payne, Mr. S. Spokes, Dr. R. Ackerley (reply)
 —, saline waters, and the use and abuse of common salt, *Baln.* 59-83; discussion (p. 83): Dr. Solly, Dr. Mouillot, Dr. G. E. Haslip, Dr. Ackerley (reply)
- Acne, extensive, case with scars (Sir M. Morris and S. E. Dore), *Derm.* 7
 —, pathology of (A. Whitfield), *Path.* 172
 —, treatment by application of vacuum electrode in static electricity, *Electr.* 85, 86
 — — — by electro-vaccination, *R.S.M.* 154
 — — — with keloids (E. G. G. Little), *Derm.* 107
 — — — by vaccine, *R.S.M.* 95, 184, 188
 — — — — acne bacillus, *R.S.M.* 138
 — — — —, staphylococcus, *R.S.M.* 137
 — — (pustular), treatment by vaccine, recurrences after, *R.S.M.* 144
- Acoustic aid for persons partially deaf (H. Macnaughton-Jones), *Otol.* 40
- Acromegaly, case of (J. R. Lunn), *Clin.* 53
 — — — (E. I. Spriggs), *Clin.* 149
- Actinomycosis, diagnosis, *Surg.* 67
 — of ovary, case of (E. H. Shaw), *Obst.* 146
- ADAMS, F. S., and HERTZ, A. F.—Formation of cholesterol gall-stones containing typhoid bacilli within sixty-eight days of the onset of typhoid fever, *Clin.* 169; discussion (p. 171): Mr. G. Wright
- ADAMS, J. E.—Case of thoracoplasty for very large empyemata, *Clin.* 90; discussion (p. 91): Mr. A. Pearce Gould, Mr. C. H. Fagge, Professor W. Osler, Mr. Arthur Barker, Mr. Adams (reply)
- ADAMSON, H. G.—Case for diagnosis, *Derm.* 55
 —, case of lichen planus annularis, *Derm.* 67; discussion (pp. 67, 68): Dr. MacLeod, Dr. Whitfield, Dr. J. J. Pringle
 — — — case of lichen simplex chronicus of Vidal, *Derm.* 41
 — — — of onychia sicca syphilitica, *Derm.* 43; discussion (p. 44): Dr. Pringle, Dr. Whitfield, Dr. MacLeod, Dr. Adamson (reply)
 —, discussion on case of lichen planus in a diabetic, *Derm.* 46, 47
 — — — of pigmented moles in a female child, *Derm.* 31
 — — — on two cases for diagnosis, *Derm.* 72
 —, elephantiasis associated with tertiary syphilis, *Derm.* 81; discussion (p. 83): Dr. MacLeod, Dr. Pringle
 —, keratosis palmaris et plantaris associated with generalized ichthyosis, *Derm.* 83
 —, linear naevus with combined verrucose and vascular elements, *Derm.* 103
 —, multiple keloid associated with "neurotic excoriations" of "dug-out" type of Colcott Fox, *Derm.* 42; discussion (p. 42): Dr. Pringle
 —, multiple scars simulating leucoderma syphiliticum, *Derm.* 1
 —, small follicular syphilide of the corymbose type, *Derm.* 1
 —, two cases of chronic superficial dermatitis in patches with symmetrical distribution, *Derm.* 65
- ADDISON, O. L.—Congenital enlargement of one limb occurring in brother and sister, *Child.* 43; discussion (p. 44): Dr. F. Parkes Weber, Mr. Lockhart Mummery
- Addison's disease, dermic pigmentation in, dependent on disturbance of suprarenal glands, *Baln.* 11
 — — —, symptoms not present in case of extensive yet incomplete fibro-caseous disease of both suprarenal capsules (H. French), *Path.* 93
- Adeno-carcinoma? large solid ovarian tumour (A. E. Giles), *Obst.* 51

- Adenoids, bacteria isolated from, R.S.M. 215
 —, growths in nasopharynx resembling, suggesting malignant disease, cases of (J. Dundas Grant), Laryng. 72, 73
 —, nature of, R.S.M. 215
 —, removal of, for gastric catarrh, Surg. 202
 —, — under anaesthetics, danger of, Anaesth. 60, 61
 —, treatment by vaccines, R.S.M. 215
 — and lymphatism, Anaesth. 44
- Adenoma of cervix of uterus, malignant, cystic (J. B. Hellier), Obst. 100
 — of liver, amyloid degeneration in (S. G. Shattock), Path. 153
 —, malignant, of liver, different terms by which known, Path. 151
 —, —, primary bile-producing tumour of liver (F. Parkes Weber), Path. 147
- Adenomata, cystic, Odont. 141
- Adenomyoma (tuberculous), definition of, Obst. 240
 — of Fallopian tube with tuberculous salpingitis (J. Inglis Parsons and B. Glendining), Obst. 238
 — of vaginal wall (T. G. Stevens), Obst. 57
- Adiposis dolorosa, cases of (G. McMullan, A. M. Gossage), Clin. 55, 58
 —, —, connexion with menopause, Clin. 59, 61
 —, —, etiology of, suggested, Clin. 59
 —, —, treatment by thyroid gland extract, Clin. 59, 60
- Adolescence, nitrous-oxide gas followed by ether, routine anaesthetic for, Anaesth. 66
- Adults, acute empyema in, following acute pneumonia or pleurisy, prognosis, Surg. 55-61
- Africa (North), and Egypt, influence of climate of, on disease (H. E. L. Canney), Baln. 115
 — (South), transmission of typhoid fever by flies in, Epid. 263
- Agglutination, discovery of, R.S.M. 4
 — of bacteria, difficulty in determining opsonic index owing to, R.S.M. 121
 — of streptococcus in case of malignant endocarditis, R.S.M. 118
- Agglutinins, production of, R.S.M. 81, 92
- Agricultural labourers, condition of, favourably compared with that of casual labourers in towns, Epid. 6
- Ainsworth, H. H., transmission of typhoid fever by house-flies, quoted, Epid. 215
- Air-passages, exclusion of, from alimentary canal after total excision of larynx, Clin. 119, 120
- Air-swallowing, case of (T. R. Whipham), Child. 151
- Air-temperature, course and interaction of, Epid. 163
 —, effect on flies, Epid. 161
 —, relation of cases of and deaths from summer diarrhoea to, Epid. 153
- Albers-Schönberg, treatment of fibroids by X-rays, quoted, Obst. 25
- Albumin, absorption of, experiments as to, Therap. 139
- Albuminuria, accompanying oxaluria, Med. 22
 —, nephritic, not increased by egg-albumin in diet, Therap. 139
 —, question of, in prognosis of chronic nephritis, Therap. 147
 —, transient, common after severe exercise without excess of albuminous diet, Therap. 139
 —, with rheumatoid arthritis, and with great enlargement of liver (W. P. Herringham), Clin. 61
- Alcohol cause of pauperism, Epid. 9
 —, ill-effects among poorest classes, Epid. 6, 7
 — on casual labourers, Epid. 6, 7
 —, injection of, into left superior laryngeal nerve, for relief of tuberculosis of larynx, with extreme odynophagia, Laryng. 152
 —, —, in treatment of dysphagia in tuberculous laryngitis, Clin. 201
 —, — of trigeminal neuralgia of left first and second divisions (W. Harris), Clin. 200
 —, weekly expenditure on, among working classes, Epid. 9
 —, see also *Intemperance*
- Alcoholics, addition of oxygen to nitrous oxide when administered to, Anaesth. 68

- Alcoholics, anæsthetization of, difficulties in, *Anæsth.* 67
 ———, leg tremor during, *Anæsth.* 68
 ———, posture in, *Anæsth.* 69
 ———, rapid in induction, important, *Anæsth.* 67, 68
 ———, reflexes dangerous in, *Anæsth.* 68
 ———, should be prolonged, *Anæsth.* 68
 ———, should be tranquil, *Anæsth.* 68
 ———, danger of ether to, *Anæsth.* 69
 Alcoholism as cause of hypersecretion, *Surg.* 181
 ———, fats in treatment of, *Med.* 33, 34
 ———, vaccine treatment of pneumonia successful in spite of, *R.S.M.* 71, 168
 ALDRICH-BLAKE, Miss.—Discussion on fibromyoma and pregnancy, *Obst.* 56
 ALDRIDGE, N. E.—Discussion on treatment of nævi by electricity, cautery and refrigeration, *Electr.* 50
 ALEXANDER, WILLIAM.—Extensive retroperitoneal hæmatocele complicating a large uterine fibroid: operation: cure, *Obst.* 139; discussion (p. 142): Dr. Macnaughton-Jones
 Alexander's operation for shortening round ligaments in treatment of uncomplicated prolapse of uterus with retroflexion, *Obst.* 228
 Alimentary canal, exclusion of air-passages from, after total excision of larynx, *Clin.* 119, 120
 Alkaptonuria in pityriasis rubra, *Derm.* 61
 ALLBUTT, Sir T. CLIFFORD.—Fatal case of myasthenia gravis, quoted, *Anæsth.* 47
 ———, on the teaching of therapeutics in the hospital wards, *Therap.* 1; discussion (p. 7): Professor Osler, F.R.S., Dr. H. Sainsbury, Dr. Calvert, Dr. R. Hutchison, Professor Dixon, Dr. Beddard, Sir Dyce Duckworth, Dr. Hackney, Professor Cushny
 Allen, negative phase of opsonic index, quoted, *R.S.M.* 191
 Allingham, H. W., and Ogle, C., operation on pericardium by gastric incision, quoted, *Med.* 91
 Alopecia areata (P. S. Abraham), *Derm.* 55
 Alternative action of vaccines, *R.S.M.* 195
 Alternating current, technique in gynecological applications, *Electr.* 34
 Amaurosis, see *Idiocy (amaurotic)*
 Amboceptors, production of, *R.S.M.* 163
 Ambulances for conveyance of scarlet-fever cases, aseptic condition, *Epid.* 82
 Amenorrhœa, treatment by electricity, *Obst.* 20, 27
 American Gynecological Society, statistics of placenta prævia, *Obst.* 169
 American Medical Association, action with regard to patent remedies, *Therap.* 99
 Amersham, medical history of, *Baln.* 38
Amœba coli, abscess of the ovary probably due to infection by (H. Williamson and J. D. Barris), *Obst.* 152
 Amputation of diseased portion in treatment of tuberculosis of epiglottis (H. Tilley), *Laryng.* 32
 Amyotonia (myatonia) congenita (A. E. Naish), *Neur.* 95
 Amyloid degeneration in adenoma of liver (S. G. Shattock), *Path.* 153
 ——— ——— ——— histology, *Path.* 155, 159
 Anæmia, choice of anæsthetic for subjects of, *Anæsth.* 70
 ———, fats in treatment of, *Med.* 33, 34
 ———, in gastric ulcer, *Therap.* 135, 136, 137
 ——— (pernicious), pigmentation of circumoral skin and of buccal mucosa in (H. D. Rolleston), *Clin.* 9
 ———, sequel to case (H. D. Rolleston), *Clin.* 216
 ———, necropsy, *Clin.* 216-218
 ——— or septic, in relation to condition of teeth, *Odont.* 99
 Anæsthesia dolorosa, *Neur.* 87
 ——— of fifth nerve and hemianæsthesia of same side, caused by pontine thrombosis (H. Campbell Thomson), *Neur.* 79
 ——— (local or general), question of choice in laryngo-fissure, *Clin.* 118, 120
 ——— (spinal), application in irreducible intussusception in children, *Child.* 175

- Anæsthesia (spinal), myxofibroma of ovary removed under (W. W. H. Tate), Obst. 73
 ———, see also *Magnesium sulphate solution, intraspinal injections*
 ———, total, in case of syringomyelia (W. Harris), Neur. 101
 Anæsthetic, cardiac and respiratory failure under, associated with engorgement of thyroid gland, Anæsth. 45, 55
 ———, choice of, conditions upon which dependent, Anæsth. 64
 ———, employment of, to determine cranial and pelvic relationships, Obst. 127
 Anæsthetics, administration of, danger of removal of adenoids under, Anæsth. 60, 61
 ——— failure of respiration under, Anæsth. 60
 ——— of, in cases of lymphatism, Anæsth. 58, 59
 ———, deaths under, in cases of lymphatism, Anæsth. 23-30, 41, 42, 58, 59
 ———, maniacal condition after passing out of anæsthetic state, Anæsth. 67
 Analgesia (local and spinal), case of prolapse of uterus with severe cardiac disease operated on by (J. Inglis Parsons and T. C. Clare), Obst. 104
 Analgesin, Therap. 86
 Anaphylaxis, experiments on, administration of ether in, Obst. 9
 Anastomosis, lateral, compared with end-to-end method, Surg. 141
 ———, in intussusception, Child. 169
 Anatomy (clinical, normal), revival of dorsal method in, Med. 214
 ANDERSON, L. GARRETT.—Specimen from a case of carcinoma of the ovary, Obst. 72; discussion (p. 72): Dr. Macnaughton-Jones
 ANDREWS, HENRY RUSSELL.—Carcinoma of the cervix of a prolapsed uterus in a patient aged 86, with a contact carcinoma on one of the labia, vaginal hysterectomy, Obst. 161; discussion (pp. 163-164): Dr. H. Macnaughton-Jones, Dr. McCann, Dr. Blacker, Dr. Andrews (reply)
 ———, case of myomectomy during pregnancy, Obst. 164; discussion (p. 166): Dr. Herman, Dr. H. Spencer, Dr. McCann, Dr. Andrews (reply)
 ———, fibroid of the vaginal wall, Obst. 129
 Aneurysm (arterio-venous), with exophthalmos, following injury to head (F. S. Eve), Clin. 77
 ——— (cirroid), see *Angioma, pulsating*
 ——— of descending thoracic aorta, diagnosis of, clinical indications establishing, Clin. 125-127
 ——— (popliteal) in both legs cured by the Matas obliterative operation (H. M. Rigby), Clin. 131
 ——— (thoracic), in boy (A. S. MacNalty), Clin. 35
 Angioma, pulsating (angioma racemosa, cirroid aneurysm), of right auricle (G. Wilkinson), Otol. 35
 Angioneurosis, cases of (J. Galloway), Clin. 106
 ——— (W. E. Wynter), Clin. 105
 ———, pain in extremity, Clin. 106
 ———, telangiectatic state, Clin. 106
 ———, treatment, Clin. 106
 ———, condition limited to women, Clin. 107
 ———, similarity of condition to Raynaud's disease, Clin. 107
 Angioneurotic oedema, family history of, associated with scleroderma of extremities (T. D. Savill), Clin. 39
 Angio-sarcoma of pineal body, case quoted, Neur. 70
 Animals, experiments on, with control series, in determination of value of vaccine therapy, R.S.M. 140
 "Angled fortification spectrum," Neur. 11
 Ankle, tibio-calcaneal resection at, with amputation of foot (C. G. Watson), Clin. 133
 Ankylosis, bony, Baln. 88, 91
 ——— (partial) of both hips with bending of bones of both legs (S. Hastings), Clin. 95
 Antibacterial substances, inoculation in different places, summation effect, R.S.M. 2, 38
 Antibodies, formation of, R.S.M. 66, 67, 92, 146, 161
 ———, difficult in glanders, R.S.M. 66, 67
 ———, production of, Therap. 104

- Antibodies, transferable from one individual to another, not forthcoming. Therap. 77
- Anti-cancer serum, fallacy of, Therap. 72
- Anti-febrin, misleading term, Therap. 88
- Antigen, R.S.M. 163
- Antikamnia, analysis of, Therap. 85
- , quack remedy, Therap. 85
- Antiseptic applications in scarlet fever, Epid. 79
- , —, —, chief value in mild or recessive cases, Epid. 79
- surgery, knowledge of bacteriology necessary to carry out, R.S.M. 3
- Antiseptics, continuous inhalations of, in treatment of incipient pulmonary tuberculosis (D. B. Lees), Therap. 17
- Anti-serum in treatment of cerebrospinal meningitis, R.S.M. 85, 86
- Antisymphilitic remedies, uselessness and injuriousness in general paralysis and true tabes, Neur. 49
- Antithyroid preparations, action of (W. Edmunds), Path. 100
- Antitoxin, production of, R.S.M. 163
- , see also *Diphtheria*, treatment by antitoxin
- immunity, paradox in, R.S.M. 197
- in treatment of tetanus, curative, Med. 40, 53
- , —, —, in conjunction with magnesium sulphate, Med. 40
- , —, —, prophylactic, Med. 40
- Antitoxins and toxins carry opposite electrical charges, R.S.M. 156
- Antitrypsin, production of, R.S.M. 163
- Antrum, mastoid, microscopical sections through, in fatal case of scarlet fever demonstrating *Streptococcus conglomeratus* in situ (S. R. Scott), Otol. 18
- , maxillary, invasion of, by suppurating dental cysts; two cases (H. Tilley), Odont. 115
- and floor of left nasal fossa, traumatic (post-operative) perforation through hard palate communicating with (L. H. Pegler), Laryng. 146
- (left) removal of rapidly growing soft fibroma from posterior wall of, by modified Rouge's operation (H. Tilley), Laryng. 96
- Anus, see *Pruritus, vulvar and anal*
- Aorta, stenosis, congenital (?) case of (A. Manuel), Child. 73
- (thoracic, descending), aneurysm of, diagnosis, clinical indications establishing, Clin. 125-127
- Aortic regurgitation, thud audible by stethoscope in arteries, Med. 211
- Apathy, mental, in cases of tumour of pineal body, Neur. 72
- Aperient, sodium chloride as, Baln. 79, 84
- Apes, syphilitic infection between, Neur. 44
- Apex-beat, rise in position in pericardial effusion, Med. 58, 101
- Aphasia in cerebral syphilis, Neur. 52
- (motor, transient), with recurring left-sided convulsions in left-handed woman (J. Taylor), Neur. 17
- Apocynamarin, Therap. 46
- Apocynin, Therap. 39
- Apocynin, Therap. 39
- Apocynum, Therap. 38
- Apostoli, introduction of electro-therapeutics into gynaecology, Electr. 4, 5, 25, 27
- , treatment of uterine tumours by electrolysis scientific, Electr. 4, 5
- Apparatus for administration of ether by purely open method (R. H. Hodgson), Anaesth. 16
- for continuous proctoclysis (H. J. Paterson), Obst. 81
- Appendages (uterine) and appendix, matting of, pain caused by, Obst. 7, 8
- , diseases of, associated with colitis, pain due to, Obst. 8
- Appendicectomy, for gastric symptoms, Surg. 187, 200
- , need of, when indicated, Surg. 207, 208
- Appendicitis in children, Surg. 202

- Appendicitis, diagnosed as gastric or duodenal ulcer, Surg. 188
 —, diagnosis, bacterial, R.S.M. 68
 — of duodenal ulcer from, Surg. 115
 —, by dorsal percussion in iliac region, Med. 238
 — following gastritis, Surg. 202
 —, gastric hypersecretion cause of liability to, Surg. 181
 —, hæmatemesis in, Surg. 195, 196
 " — larvata," Surg. 187
 —, Meckel's diverticulum containing calculi and producing colic and simulating, Clin. 12, 15
 — (suppurative), symptoms of, Surg. 184
 —, treatment by vaccine, case unsuccessful, R.S.M. 185
 Appendicostomy, effect on gastric hypersecretion, Surg. 190
 — for treatment of hæmorrhage in pneumococcal colitis with hyperpyrexia (H. E. Bruce-Porter, J. P. Lockhart Mummery, and W. Hale White), Clin. 48
 " Appendicular colic," Clin. 12, Surg. 206
 " — gastralgia," symptoms, Surg. 194
 Appendix vermiformis, acutely inflamed, involuted into lumen of cæcum, which then formed apex of intussusception: operation: recovery (I. Back), Child. 25
 — and adnexa, matting of, pain caused by, Obst. 7, 8
 — as cause of gastric symptoms (H. J. Paterson), Surg. 187
 —, disease of, cause of hæmatemesis, Surg. 191, 207
 — —, diagnosis, gastric symptoms, Surg. 194
 — —, hyperchlorhydria in, Surg. 201
 — —, origin of gastric symptoms, Surg. 188
 —, latent disease of, connexion with gastric hypersecretion (W. S. Fenwick), Surg. 177
 — dyspepsia, Surg. 115
 Appetite in duodenal ulcer during stage of pain, Surg. 71
 —, excessive, accompanying case of tumour of pituitary body, Neur. 72
 Apraxia, following syphilitic infection, Neur. 54
 Argyll-Robertson pupil, physical sign common to tabes dorsalis and general paralysis, Med. 43, 47
 Argyria, case of (F. P. Weber and R. H. Norman), Derm. 75
 Armlet, simultaneous employment of stethoscope with, to combine auscultatory and tactile methods of reading arterial pressure (G. Oliver), Med. 207
 — and manometer, use of in measuring arterial blood-pressure, Med. 208, 209
 ARMOUR, D.—Discussion on excision of entire tongue, Laryng. 138
 ARMSTRONG, H. G.—The case-incidence in nine epidemics of measles at a public school, with notes on the pre-eruptive symptoms, Epid. 54; discussion (p. 65): Dr. Goodhart, Dr. T. Thomson, Dr. Parsons, Dr. F. M. Turner, Dr. Biernacki, Dr. Buchan, Dr. M. Richards, Dr. Crookshank, Dr. Thresh, Mr. Armstrong (reply), Dr. Milne (reply)
 ARMSTRONG, W.—Discussion on arthritis deformans, Baln. 111
 Army, British, see also *India, British Army in*
 — —, syphilis in, Surg. 225
 — —, treatment of, Surg. 223
 — —, venereal diseases in, Advisory Board Reports on, Surg. 225
 Arsacetin in treatment of syphilis, Surg. 217, 225
 — — — followed by blindness, Surg. 225
 Arsenic, application of, in treatment of lupus of nose and throat, Laryng. 87
 — — — — of syphilis, Surg. 216, 231
 Arterial pressure (systolic and diastolic), auscultatory and tactile methods of reading, combined (G. Oliver), Med. 207
 Arteries, atheroma of, supposed remote bacterial origin of, R.S.M. 13
 —, blood-pressure in, auscultatory method of reading described and demonstrated, Med. 209, 210

- Arteries, blood-pressure in, increase of, due to excessive ingestion of sodium chloride, Baln. 74
 ——— (systolic and diastolic), tactile and visual methods of reading, limitations of, Med. 207, 208
 ——— (carotid, external), temporary ligature of both, and laryngotomy; fibrosarcoma of nose removed after (D. Harmer), Clin. 122
 ——— (cerebral), hæmorrhage from, see *Hæmorrhage, cerebral*
 ———, syphilitic thrombosis of (H. Head), Neur. 30
 ——— thud audible in, by stethoscope in aortic regurgitation, Med. 211
 Arterio-sclerosis and chronic pancreatitis, Med. 174, 175
 ———, importance of examination of teeth in cases of, Odont. 95
 ——— and osteo-arthritis, Baln. 89
 ———, association with chronic gastric ulcer, Therap. 134
 Arteritis obliterans, chronic, of lower extremity, associated with phlebitis (F. Parkes Weber), Clin. 97
 ———, intermittent claudication due to, Clin. 96
 ———, universal, with syphilitic meningitis, Neur. 51
 Artery (coronary), ligature of, third step in operation for removal of lymphatic area connected with stomach, Surg. 152
 ——— (hepatic), ligature of branches of, fourth step in operation for removal of lymphatic area connected with stomach, Surg. 152
 ——— (left posterior cerebellar), thrombosis of, followed by severe trigeminal neuralgia in analgesic facial area (W. Harris), Neur. 81
 ——— (pulmonary), atresia, complete, shown in specimen of congenital malformation of heart (A. C. D. Firth and E. I. Spriggs), Child. 49
 Arthritis and endocarditis, Therap. 102, 104
 ——— deformans (R. Llewelyn Jones), Baln. 85
 ——— (reply), Baln. 112
 ———, discussion on, Baln. 99
 ——— (gonococcal), auto-inoculation experiments in, R.S.M. 25
 ———, "rheumatic nodules" in probable case of (H. Morley Fletcher), Clin. 107
 ———, treatment by vaccine, R.S.M. 55-56, 183, 188, 189
 ——— (gonorrhœal) in women, Baln. 111, 112
 ——— (gouty), connexion with inadequate metabolism, Baln. 8
 ——— (— and rheumatoid), accompanied by, and due to, thyroid insufficiency, Baln. 8, 9
 ———, amenability to balneary therapeutics, Baln. 8, 9
 ———, mutually exclusive, Baln. 8
 ——— (infective), Baln. 87, 92
 ——— (rheumatoid), Baln. 86, 91, 94
 ———, comparison of acute nephritis with, Baln. 102
 ———, constitutional disturbances in, Baln. 101
 ———, diagnosis by Röntgen rays, Baln. 100, 102
 ———, importance of examination of teeth in, Odont. 95, 99
 ———, influenced beneficially by climate of Egypt and North Africa, Baln. 126-128
 ———, points of distinction from osteo-arthritis, Baln. 100, 101
 ———, relieved by static wave current, Electr. 104
 ———, with albuminuria, and with great enlargement of the liver (W. P. Herringham), Clin. 61
 ———, changes in joints and muscular wasting of, as due to irritative lesions in spinal cord, Baln. 103, 106
 ———, relation of uterine disorders to, Baln. 105
 ———, treatment by counter-irritation, Baln. 109-111
 ———, with enlargement of lymphatic glands and spleen (E. I. Spriggs), Clin. 15
 ———, ———, see also *Still's disease*
 ———, see also *Osteo arthritis, metabolic*
 ——— (tuberculous), treatment by vaccine, R.S.M. 129-136

- Aryl-arsenates in treatment of syphilis, Surg. 216
 ———, blindness following, Surg. 233, 244
 ———, value discussed, Surg. 218, 231
 ———, see also *Arsacetin*; *Atoxyl*; *Orsudan*; *Mercury, atoxylate of*; *Soamin*
 ——— of trypanosomiasis, justifiable and imperative, Surg. 219
 Arytenoid cartilage, left, ulcer of doubtful nature on (H. Betham Robinson), Laryng. 29
 ———, see also *Inter-arytenoid space*
 Ascites, chronic, connected with hepatic cirrhosis, and treatment by omentopexy and peritoneal drainage, patient shown a few months after (F. P. Weber), Clin. 80
 ———, disappearance of, in case of alcoholic cirrhosis after paracentesis and laparotomy (F. P. Weber and E. Michels), Clin. 167
 ———, new physical test for, Med. 220
 Askanazy, on premature development in children the subjects of tumour of pineal body, Neur. 75
 Aspiration, in diagnosis of gastric disorders, Surg. 183
 ——— for heart stoppage, value of, Anæsth. 5
 Aspirin, synonym for acetyl salicylic acid, Therap. 87, 101
 Assouan as a health resort, Baln. 124
 Asthenics, choice of anæsthetics for, Anæsth. 66
 Asthma, beneficial influence of climate of Egypt and North Africa on, Baln. 126
 ———, case of; improvement after nasal treatment (D. McKenzie), Laryng. 116
 Asylums, prevalence of influenza, dysentery or pneumonia in, followed by death of number of general paralytics, Neur. 49
 Ateleiosis in man aged 42; physical development said to have been arrested at age of 9 years (F. Parkes Weber), Child. 143
 Athletes, transient albuminuria in, Therap. 139
 Atmospheric electricity (G. Mahomed), Baln. 49-58
 ———, influence of sun-spots on, Baln. 54
 ———, ionization in, Baln. 53
 ———, potential, Baln. 51, 55-58
 Atoxyl, date of first preparation, Therap. 88
 ——— injections in treatment of syphilis, Surg. 217, 231, 237
 ———, blindness following, Surg. 225, 231
 Atrophy, olivo-ponto-cerebellar, case of (G. Holmes), Neur. 89
 Attrition, painful, case of (W. Rushton), Odont. 118
 Auditory apparatus, some features in, of a 16 mm. human embryo, shown in reconstruction model (by wax-plate method of Born) (G. J. Jenkins), Otol. 34
 ——— canal, external, cholesteatoma or keratosis obturans of, specimen (H. Todd), Otol. 81
 ——— discharges from organic disease, Neur. 5
 Auenbrugger, Leopold (1722-98), "*Inventum Novum ex Percussione Thoracis humani ut signo abstrusos interni pectoris morbos detegendi*," 1763, quoted, Med. 211, 212
 Auenbrugger's sign in pericardial effusion, Med. 58, 62
 Auer and Meltzer, experiments on monkeys to test value of magnesium-sulphate treatment of tetanus, Med. 41
 Auricle, left, dilation of, in pericardial effusion, Med. 57
 ——— right, pulsating angioma of (G. Wilkinson), Otol. 35
 Auricles, thickening of the cartilage of both (? othematoma) (D. McKenzie), Otol. 53
 Auscultation, in diagnosis of early pulmonary tuberculosis, Therap. 24
 Australia, aboriginals of, correct occlusion of teeth seen in, Odont. 156
 Auto-inoculation, R.S.M. 85, 209
 ———, experiments in gonococcal arthritis, R.S.M. 25
 ——— in phthisis, R.S.M. 25
 ——— in combination with taking of opsonic index, R.S.M. 46
 ———, induction of, R.S.M. 4
 Auto-inoculations of disease, deficient or excessive, R.S.M. 162

- Autolysin reaction, impossibility of obtaining in cancer immunization, Therap. 77
 Auto-vaccination, production or control by electrical means, R.S.M. 157
 —, see also *Electro-vaccination*
 Axis cylinders, degeneration of, after snake-venom poisoning, Neur. 112, 113
 Axolysis of nerve-fibres, Path. 25
 Axon, definition of, Path. 21
 —, fibrils composing, Path. 21, 23
 Aylesbury, medical history of, Baln. 39
- BABER, E. CRESSWELL.—Discussion on case of audible tinnitus, Otol. 1
 —, discussion on model demonstrating "back" and "belly" breathing, Laryng. 48
 — on post-operative tests in labyrinthine disease, Otol. 6
 Babinski's sign, presence of, after syphilitic infection, Neur. 96, 98
 — in syphilitic disease of spinal cord, Neur. 56, 58, 59
 Bacelli, hypodermic carbolic-acid solution treatment of tetanus, Med. 40
 Bacilli in tetanus, destruction, Med. 40
 —, generalization, Med. 40
 —, pathogenic growth in soil, Epid. 138, 139
 —, typhoid, in cholesterol gall-stones (A. F. Hertz and F. S. Adams), Clin. 169
Bacillus acidii lacti, presence of, in mucous colitis determined by auto-inoculation in combination with opsonic index, R.S.M. 46, 47
 — *acnes*, Path. 172
 —, R.S.M. 137
 —, vaccine of, in treatment of acne vulgaris, R.S.M. 138
 — (Bordet's), found in whooping-cough, R.S.M. 97
 — *bulgaricus* (lactic-acid bacillus), administration in pure cultures, Therap. 53
 —, administration in tablet form, Therap. 53, 58
 — not recommended, Therap. 60
 —, dietetic value, Therap. 61
 —, effect on pneumococcal and streptococcal invasion of intestines, Therap. 60, 62
 —, impure and defective preparations, Therap. 54
 —, indiscriminate recommendation by manufacturing chemists and milk-vendors, Therap. 51, 52
 —, limitations of use, Therap. 52, 58
 —, therapeutic application, indications for, Therap. 54, 55, 59, 60, 62
 — value (G. Herschell), Therap. 51-56
 —, see also *Lactic-acid preparations*; *Sour-milk treatment*; *Trilactine*
 — *coli communis*, indifferent definition in vaccine therapy, R.S.M. 41
 —, infection by, retroperitoneal sarcoma mistaken for, R.S.M. 142
 —, treatment with vaccine, R.S.M. 57, 104, 105, 188
 — vaccine, effects of, in case of general infection, R.S.M. 213, 214
 — *enteritidis sporogenes* and summer diarrhoea, Epid. 133
 — (Friedländer's), infection causing suppuration of frontal sinus, treatment by vaccine, R.S.M. 189
 —, vaccine from, in treatment of case of subacute rheumatism, R.S.M. 177, 178
 — *fusiformis*, Odont. 30, 31, 35
 — *malleti*, opsonic index to, R.S.M. 42, 43; see also *Glanders*
 — *maximus buccalis*, Odont. 30
 — *neoformans*, vaccines of, in treatment of cancer, Therap. 78
 — *prodigiosus*, suspension method of preparation, Surg. 8
 —, toxins of, Therap. 79
 — and *Streptococcus erysipelas*, mixed toxins of, in treatment of inoperable carcinoma (W. B. Coley), Surg. 1-44

- Bacillus prodigiosus* and *Streptococcus erysipelas*, mixed toxins of, preparation, Surg. 7
 ———, method of preparation, Surg. 8
 ———, see also under *Toxins (mixed)*
 ——— *proteus*, invading wound of tuberculous ulceration, R.S.M. 21
 ——— *pseudo-tuberculosis rodentium*, R.S.M. 162, 163
 ——— *pyocyaneus*, infecting organism in septic perichondritis, Otol. 71, 72
 ———, presence in middle-ear disease, R.S.M. 160
 ———, in urine from supposed case of muscular rheumatism, R.S.M. 178
 ———, vaccine of, effect of digestion on, R.S.M. 91
 ——— *segmentosus coryzae*, indifferent definition in vaccine therapy, R.S.M. 40
 ——— *septus*, present in cultures from hay fever, R.S.M. 16
 ——— *xerosis*, Odont. 35
- BACK, IVOR.—Case of acute inflammation in an appendix, involution into the lumen of the caecum, which then formed the apex of an intussusception; operation; recovery, Child. 25; discussion (p. 26): Mr. Lockhart Mummery, Mr. Back (reply)
- Back-breathing, advantages of, Laryng. 43
 ———, characterization of, and results, Laryng. 42, 43
 ———, definition, Laryng. 42
 ———, demonstration of, by model, Laryng. 46
 ———, variation in effect on stresses, strains, and frictions in throat and larynx, illustrated by model (R. H. Scanes Spicer), Laryng. 41
- Backmann, cream and butter in treatment of gastric disorders, Med. 26;
- Bacteria, agglutination of, difficulty in determining opsonic index owing to, R.S.M. 121
 ———, immunity reactions to, Therap. 78
 ———, infection by, in scarlet fever, Epid. 80
 ———, inoculation of killed and living, comparative consequences discussed, Therap. 78
 ———, intestinal, fat-splitting action, Med. 191, 193, 196
 ———, introduction from duodenum into pancreatic duct in dogs producing chronic pancreatitis, Path. 83, 90
 ———, relation of saponified to unsaponified fats in faeces in diseases of pancreas determined by distribution and activity of, Med. 193
 ———, isolated from adenoid growths, R.S.M. 215
 ——— (killed), action of digestive ferments on, R.S.M. 93
 ———, oral administration, R.S.M. 92
 ———, amount absorbed bears no constant relation to amount swallowed, R.S.M. 93
 ———, proof of, causal relationships before use as vaccines, R.S.M. 97
 ———, see also *Mouth, diseases of, bacterial*
- Bacterial infection common to every disease directly or remotely, R.S.M. 13, 14
 ———, individual susceptibility to different kinds of, R.S.M. 19
 ———, resisting power of organism to, complete or partial evaluation, R.S.M. 29, 30
 ———, infections, classes of, R.S.M. 22
 ———, diagnosis by physical signs, uncertain, R.S.M. 5
- Bactericidal agents in brush-discharges, Electr. 84
- Bacteriologists, discoveries in infective diseases, the work of, R.S.M. 77, 78
 ———, ignorance of clinical medicine among, R.S.M. 113
 ———, indebtedness of clinicians to, R.S.M. 76, 77
 ———, limitations to vaccine therapy, claimed by, R.S.M. 12, 19, 20
 ———, position of, R.S.M. 78
 ———, relations with clinicians, R.S.M. 9, 60, 61, 110, 111, 117, 143, 145, 187, 210
- Bacteriology, ignorance of clinicians of, R.S.M. 9
 ———, knowledge of, essential to clinicians, R.S.M. 113
 ———, throughout medical profession, R.S.M. 5
 ———, of, necessary in antiseptic surgery, R.S.M. 3
 ———, of pericarditis with effusion, Med. 108
 ———, of purulent pericarditis, Med. 67

- Bacteriology of pyorrhœa alveolaris (J. W. Eyre and J. L. Payne), *Odont.* 29
 —, relations of recent history of medicine to, *R.S.M.* 3
 —, specialization of work in, *R.S.M.* 5, 6
 —, standards of, to determine value of vaccine therapy, *R.S.M.* 39-41
 —, study of, position of clinicians towards, *R.S.M.* 4, 102
 BADCOCK, J. H.—Discussion on sterilizable film-holder, *Odont.* 89
 —, discussion on susceptibility of teeth to dental caries, *Odont.* 82
 BAILLY, rupture of tunica vaginalis in hydrocele, *Surg.* 166, 173
 BAKER, C.—Discussion on acute pemphigus of larynx, *Laryng.* 22
 Bakers, danger of allowing subjects of tuberculosis to be employed as, *Clin.* 119
 BALDWIN, H.—Discussion on absorption of teeth, *Odont.* 18
 —, discussion on exostoses of the mandible, *Odont.* 26
 BALLANCE, C. A., M.V.O.—Case of fracture of the base of the skull: operation: recovery, *Clin.* 82
 —, discussion on radical operation for cancer of pylorus, *Surg.* 161
 BALLARD, E., references to communicability of epidemic diarrhœa, quoted, *Epid.* 95
 —, report on epidemic pleuro-pneumonia, quoted to illustrate characteristic features of communicable diseases, *Epid.* 99
 —, summer diarrhœa, quoted, *Epid.* 132, 133
 Balneary therapeutics, amenability to, of group of diseases probably due to thyroid inadequacy, *Baln.* 8, 9
 Balneology, future of, *Baln.* 12
 Bamberger's sign, as denoting presence of pericardial effusion, *Med.* 87
 —, in pericardial effusion, *Med.* 59, 74
 Band, strangulation of small intestine by, in infant (J. P. Lockhart (Mummery)), *Child.* 37
 Bands between Eustachian tube and pharynx (E. A. Peters), *Laryng.* 69
 —, —, —, question whether natural or post-operative discussed, *Laryng.* 69
 BARCLAY, A. D.—Discussion on examination of intestines by X-rays, *Electr.* 123
 —, discussion on relative value of various transformers, *Electr.* 155
 —, on X-ray treatment of lymphadenoma, *Electr.* 133
 BARKER, ARTHUR E.—Case for diagnosis, ? endothelioma, *Clin.* 147; discussion (p. 148): Mr. Barker, Mr. H. Evans, Dr. H. D. Rolleston, Dr. Finzi
 — case of traumatic pancreatic cyst cured by operation and followed twelve years later by medullary leukemia, *Clin.* 121
 —, discussion on case of lymphangioma of tongue, *Clin.* 112
 —, —, —, of epithelioma of larynx shown after operation, *Clin.* 118
 —, —, —, on pyæmic knee- and shoulder-joints treated by tapping and incision, *Clin.* 121
 —, —, —, on thoracoplasty in treatment of empyema, *Clin.* 92
 —, —, —, on tibio-calcaneal resection at ankle, *Clin.* 134
 —, —, —, on treatment of stricture of rectum and other conditions by fibrolysin, *Clin.* 89
 —, —, —, on treatment of tetanus, *Med.* 53
 Barker's operation in intussusception, *Child.* 169
 Barker, Milton R., on pancreatic reaction, quoted, *Med.* 182
 Barlow, Sir T., case of foetal cretinism alluded to, *Path.* 110
 Barnes, Robert, "dysocotocia," quoted, *Obst.* 5
 BARNES, STANLEY.—Discussion on syphilis and parasymphilis of the nervous system, *Neur.* 64
 Barr, Sir J., danger of foul mouths, quoted, *Odont.* 101
 Barrier system, arrangement of cases in isolation hospitals by, *Epid.* 83
 BARRIS, J. D.—Tumour of the uterus, of doubtful nature, *Obst.* 73
 — and WILLIAMSON, H.—Abscess of the ovary probably due to infection by the *Amœba coli*, *Obst.* 152; discussion (p. 153): Dr. C. Lockyer
 Barrois, tunica vaginalis, quoted, *Surg.* 167
 BARTON, G. A. H.—Discussion on choice of anæsthetic, *Anæsth.* 73
 BARWELL, HAROLD.—Case of probable malignant disease of the larynx in a woman aged 47. *Laryng.* 36; discussion (p. 37): Mr. Atwood Thorne, Mr. de Santi, Dr. H. J. Davis, Mr. Barwell (reply)

- BARWELL, HAROLD.—Discussion on case of congenital occlusion of left posterior nares, Laryng. 92
 —, discussion on case of laryngeal growth, Laryng. 39
 — — — of "singer's node" on left cord, Laryng. 108
 — — — of tuberculosis of epiglottis treated by amputation of diseased portion, Laryng. 32
 — — — on left abductor paralysis, Laryng. 137
 — — — on lupus of nose and throat, Laryng. 87
 — — — on model of upper and lower jaws and impression of roof of mouth, Laryng. 141
 — — — on peculiar erythema of left palate, Laryng. 27
 — — — on traumatic perforation through hard palate, Laryng. 146
 —, perichondritis of thyroid cartilage of unknown origin, Laryng. 109
 —, tuberculous perichondritis of cricoid and arytaenoid cartilages, Laryng. 109
 —, ulceration of the epiglottis, probably epitheliomatous, Laryng. 145; discussion (p. 145): Dr. J. Dundas Grant, Mr. Barwell (reply)
 BASEIL, rupture of tunica vaginalis in hydroceles, Surg. 171
 BASHFORD, E. F.—The immunity reaction to cancer, Therap. 69-81
 —, incidence of cancer, quoted, Laryng. 159
 —, inhibition of growth of graft of mouse-epithelioma, quoted, Path. 129
 BATHS, stimulation of cutaneous surface by, Baln. 7, 10
 BATTEN, F. E.—Discussion on case of acute cerebral softening, Neur. 132
 —, discussion on case of cerebellar sclerosis, Neur. 91
 — — — on case of compression paraplegia: syphilitic meningitis, Neur. 22
 — — — on paralysis of right vocal cord in myotonia atrophica, Laryng. 21
 — — — on secondary growths affecting spinal roots, Neur. 129
 — — — on two cases of myotonia atrophica, Clin. 147
 —, distal type of myopathy in boy, also in several members of a family, Neur. 92, 93; discussion (p. 94): Dr. F. Buzzard, Dr. Warrington, Dr. J. A. Ormerod, Dr. G. Holmes, Dr. F. E. Batten (reply)
 —, on contraction of diarrhoea by nurses in children's hospitals, quoted, Epid. 106
 BATTEN'S frog-child, case of (T. H. Openshaw), Clin. 39
 BATTEN, G. B.—Discussion on treatment of naevi by electricity, cautery and refrigeration, Electr. 49
 BAYLY, H. W.—Demonstration by the ultra-microscope of living *Treponema pallidum* and various spirochetes, Clin. 3
 —, discussion on present position and treatment of syphilis, Surg. 238
 — — — on vaccine therapy, R.S.M. 72
 BAZIN'S disease, case of (G. W. Dawson), Derm. 27
 BEACONSFIELD, medical history of, Baln. 40
 BEARD region, hand and upper lip, ringworm affecting (S. E. Dore), Derm. 111
 BED ISOLATION system in fever hospitals, Epid. 83, 84
 — — — — —, advantages of, Epid. 84
 — — — — —, reduction of scarlet-fever occurrence-rate under, Epid. 85
 BEDDARD, A. P.—Discussion on teaching of therapeutics in hospital wards, Therap. 14
 BEDDOES, T. P.—Discussion on proprietary, patent, and secret remedies, Therap. 91
 —, discussion on the treatment of tetanus, Med. 54
 BEER-trap for capture of house-flies, Epid. 141, 142, 169
 BELFAST, typhoid fever in, causes of prevalence, Epid. 270-74
 BELLY-breathing, characterization of and results, Laryng. 43, 44, 49
 —, definition, Laryng. 42
 —, demonstration of, by model, Laryng. 46
 —, transverse axis of respiratory rotation of cricoid on thyroid cartilage in, illustrated by model (R. H. Scanes Spicer), Laryng. 41, 44, 49
 —, variation in effect on stresses, strains, and frictions in throat and larynx, illustrated by model (R. H. Scanes Spicer), Laryng. 41
 BENNETT, F. J.—Discussion on absorption of teeth, Odont. 18

- BENNETT, F. J.—Discussion on case of painful attrition, *Odont.* 120
 —, discussion on exostoses of the mandible, *Odont.* 25
 — on odontocles and other cysts, *Odont.* 158
 — on susceptibility of teeth to dental caries, *Odont.* 80, 83
 — on unilateral overgrowth of lower jaw, *Odont.* 50
 Benzoin, compound tincture of, inhalation of, in treatment of case of hoarseness, *Laryng.* 56
 Béraud, on tunica vaginalis, quoted, *Surg.* 168
 BERKELEY, C.—Treatment of puerperal eclampsia, quoted, *Obst.* 193
 — and BONNEY, V.—Leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, *Obst.* 29; discussion (p. 48): Dr. Macnaughton-Jones, Dr. Giles, Dr. Routh, Dr. Eden, Dr. Berkeley (reply)
 Bernard and Marie, cases illustrating effects of syphilitic virus on nervous system, quoted, *Neur.* 43
 BERRY, Mrs. DICKINSON.—Discussion on choice of anæsthetic, *Anæsth.* 75
 Berry, J., on prevalence of goitre in Chilterns, quoted, *Baln.* 45
 Bertrandi, rupture of tunica vaginalis in hydroceles, quoted, *Surg.* 165
 Besredka, administration of ether in experiments on anaphylaxis, quoted, *Obst.* 9
 Besredka's method of prophylactic inoculation, *R.S.M.* 85
 Betz cells, *Path.* 65
 —, changes in, *Path.* 66, 67, 69
 —, chromatolysis of, *Path.* 17
 —, fibrils of, connexions discussed, *Path.* 29
 BIERNACKI, J.—Discussion on scarlet fever: home treatment and prevention, *Epid.* 69
 BIGGS, G. N.—Case of long-continued suppuration (aural), with spontaneous cure? *Otol.* 15
 Bile, absence of, from intestine, effects, *Clin.* 84
 —, see also *Fistula (biliary)*
 Bile-pigment in feces in diseases of pancreas, *Med.* 197
 —, test employed to detect, *Med.* 198
 Bile-producing primary malignant tumour of liver ("malignant adenoma") (F. Parkes Weber), *Path.* 147
 Billroth's first and second methods of gastrectomy, results compared, *Surg.* 140, 141
 Bismuth, administration of, before examination of intestines by Röntgen rays, *Electr.* 109-124
 Black, investigations on components of teeth, *Odont.* 72
 Blackburn, Nursing Mothers' Aid Society established at, *Odont.* 6
 BLACKER, G. F.—Discussion on carcinoma of prolapsed uterus, *Obst.* 163
 —, discussion on double uterus with dysmenorrhœa, *Obst.* 242
 —, on pelvic hæmatocele of ovarian origin, *Obst.* 111
 —, on squamous-celled carcinoma of a dermoid cyst of the ovary, *Obst.* 145
 Bladder, acute coli infection of, treatment by vaccine, *R.S.M.* 73
 —, atony of, greater trouble in old age than enlargement of prostate, *Electr.* 146
 —, relieved by application of rectal electrode, *Electr.* 147
 —, with pendulous abdomen, mechanical stimulation in, *Electr.* 147
 —, cancer of, choice of operation, *Clin.* 73
 —, diseases of, diagnosis by cystoscopy, important, *Clin.* 74
 —, excision of, when to be preferred to resection, in case of growths, *Clin.* 74
 —, irritability of, in women, treatment by electricity, *Electr.* 7, 8, 11, 12, 30
 —, radiographs produced through Snook's high-tension transformer, *Electr.* 158
 —, resection of, for malignant disease, case: (J. W. Thomson Walker), *Clin.* 68
 —, importance of Trendelenburg position, *Clin.* 72
 —, technique, *Clin.* 72
 —, rhabdomyoma of (S. G. Shattock), *Path.* 31
 —, histology, *Path.* 33
 —, museum specimens, *Path.* 31, 36, 38, 39
 —, origin of growth discussed, *Path.* 39
 —, see also *Ectopia vesicæ*

- Blake, generalization of bacilli in tetanus, Med. 40
- BLAND-SUTTON, J.—Cases of rupture of tunica vaginalis in hydroceles, Surg. 173
- , leucoplakic vulvitis, quoted, Obst. 29
- , use of term "dentigerous cyst," Odont. 161
- Bleeding, see *Phlebotomy*
- Blindness due to ophthalmia neonatorum, expense to state, Surg. 225
- following injection of arsacetin, Surg. 225
- — — of aryl-arsenates, Surg. 233
- — — of atoxyl, Surg. 231
- — — of orsudan, Surg. 217
- — — of soamin, Surg. 217, 218
- Blood, calcium oxalate cannot exist in, Med. 5
- , circulation of, action of abdominal muscles on, Laryng. 48, 51
- , condition of, in case of chloroma, Child. 96
- , examination for, in faeces in diseases of pancreas, Med. 199, 200
- — of, in serous pericarditis, Med. 63
- and mucus, passage of, in intussusception, Child. 168
- , opsonic power of, discovery, R.S.M. 4
- — —, phagocytic activity dependent on, R.S.M. 164
- , test of, after severe exercise to determine extinction of bacterial infection, R.S.M. 24, 25
- Blood changes after X-ray exposures, R.S.M. 158
- Blood-corpuscles, red, protective powers of, Baln. 11
- Blood-culture to procure autogenous pneumococcus vaccine, R.S.M. 166
- Blood-fluids, action on leucocytes, method for studying (H. Chambers), Path. 73
- Blood-plasma, condition of, influenced by thyroid gland secretion, Baln. 7, 8
- Blood-pressure, see *Arteries, blood-pressure in*
- Blood-serum, examination by Wassermann reaction during pregnancy, Neur. 121
- , proportion of sodium chloride in saline waters compared with that contained in, Baln. 78, 79
- of idiots, examination by Wassermann reaction (H. R. Dean), Neur. 117
- Blue patches on buttocks and lumbar regions, case of (F. Langmead), Child. 133
- BLUMFELD, J.—Discussion on "lymphatism," Anæsth. 49
- Board of Education, duty to working classes, Epid. 40
- Boas, fats in treatment of gastric disorders, quoted, Med. 25, 26, 27
- Body-weight and gastric cancer, Surg. 159
- Boidin and Weil, P., acute meningitis during secondary syphilis, quoted, Neur. 37
- Boils, treatment by vaccine, R.S.M. 65, 95, 103, 125, 188
- — —, recurrences after, R.S.M. 144
- Boldyreff, fats in treatment of gastric disorders, quoted, Med. 30, 32
- BOLTON, CHARLES.—Some points in the treatment of gastric ulcer, Therap. 117-133; discussion (p. 133): Professor Dixon, Dr. E. I. Spriggs, Dr. Hertz, Dr. Langdon Brown, Dr. Herbert French, Dr. Bolton (reply)
- BOLTON, J. S.—The relief of symptoms of prostatic obstruction by electrical treatment, Electr. 141.146; discussion (p. 146): Dr. Sloan, Dr. Primrose Wells, Dr. Howard Humphris, Dr. Bolton (reply)
- Bone, injury of, virulent sarcoma due to, Surg. 11
- , sarcoma of, diagnosis from cystic disease, Clin. 195
- Bone-marrow, condition found at necropsy in case of pernicious anæmia with pigmentation of circum-oral skin and buccal mucosa, Clin. 218
- Bones (fœtal), grafting into pregnant does, Path. 132
- — — of, attempts to produce chondromatous or osteomatous growths by (S. G. Shattock, C. G. Seligmann, and L. S. Dudgeon), Path. 127-140
- — — into young rabbits, Path. 131
- — — of, technique, Path. 130

- Bones, intra-cartilaginous growth diminished in cachexia strumipriva, Path. 106
- in cretins, Path. 106
- (long), in dachschund, histological details of ossification, Path. 108-110
- , in male Belgian hare, effect of castration on, Path. 104, 105
- , sarcoma of, use of mixed toxins in, Surg. 12-16
- , normal growth of, aided by internal secretion of thyroid gland, Path. 138, 139
- of pig, effect of feeding with madder on, Odont. 80, 83
- BONNEY, VICTOR.—A secondary implantation teratomatous cyst, Obst. 151
- and BERKELEY, C.—Leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, Obst. 29; discussion (p. 48): Dr. Macnaughton-Jones, Dr. Giles, Dr. Routh, Dr. Eden, Dr. Berkeley (reply)
- Boobbyer, P., on typhoid and diarrhœal death-rates in Nottingham and Leicester, Epid. 111
- Booth, C., method of estimation of poverty in London, Epid. 4, 14, 15
- Bordet's bacillus found in whooping-cough, R.S.M. 97, 98
- Bordet and Gengou on complement-deviation, quoted, R.S.M. 163
- Bordier, treatment of uterine fibroids by radium, Electr. 71
- Born, wax-plate method of, used in reconstruction model showing auditory apparatus of 16 mm. human embryo (G. J. Jenkins), Otol. 34
- Bossi's mechanical dilatation of cervix in treatment of puerperal eclampsia, Obst. 204, 205
- Bottle-feeding of infants, Odont. 7
- Bovine tuberculous mastitis, see *Mastitis*, bovine tuberculous
- Bowlby, A. A., origin of osteo-arthritis, quoted, Baln. 90
- Box, C. R.—Discussion on pericardial effusion, Med. 95
- , discussion on specimen of hypertrophic stenosis of pylorus, Child. 100
- and BUTLER, G. G.—Statistics of pericarditis, with effusion, from St. Thomas's Hospital, Med. 104
- Boy, exophthalmic goitre (Graves' disease) occurring in (E. Pritchard and S. Stephenson), Child. 73
- , exostosis of right external meatus in (H. Todd), Otol. 76
- BOYD, Mrs. F. N.—Discussion on abdominal hysterectomy for necrosed and suppurating subperitoneal fibroid, Obst. 65
- , discussion on case of cystic tumour of right broad ligament, Obst. 71
- BOYD, STANLEY.—Case of unilateral overgrowth of lower jaw, Odont. 47; discussion (p. 49): Mr. Colyer, Mr. Mummery, Mr. Sturridge, Mr. Mountford, Mr. Lloyd Williams, Mr. F. J. Bennett, Mr. Boyd (reply)
- Brachial plexus, neuritis of, case of, further notes (W. P. Herringham), Clin. 28
- Brain, abscess of, complicating lung diseases, Surg. 67
- , cortex of, chromatolysis of cells of, from case of electric shock, Path. 145
- , hæmorrhage in, from case of electric shock, Path. 142
- , parts from which sections were made from case of transverse lesion of lower cervical region of spinal cord, Path. 71
- , changes in cells of central area, Path. 70
- , grey matter of, see *Glia cells*
- , hemisphere left, hemiatrophy of (A. C. D. Firth and J. C. G. Ledingham), Child. 53
- , ——— histological report on section of leptomeninges and underlying brain matter from upper parietal region, Child. 54
- , histological changes in specimen from case of electric shock of 20,000 volts (F. W. Mott, F.R.S., and E. Schuster), Path. 140
- , malignant tumour of, olfactory discharges due to, Neur. 2
- , sarcoma of, large-celled specimen (J. T. Leon), Child. 160
- , softening of, acute, possibly due to venous thrombosis (W. Harris and B. H. Spilsbury), Neur. 130
- , syphilitic vascular lesions of, effects in two cases, Neur. 30, 31
- BRAINE, C. CARTER.—Discussion on lymphatism, Anæsth. 45
- Braxton Hicks, method compared with Cesarean section in placenta prævia, Obst. 171-173

- Breast, cancer of, treatment by radium, case of, further report (N. S. Finzi and E. H. Shaw), Clin. 1
- , carcinoma of, physiological capacity of cells, Path. 162, 163, 164
- , cystic degeneration of, supposed toxic origin of, R.S.M. 13, 14
- , gummæ of, simulating malignant diseases (H. J. Paterson), Clin. 82
- , streptococcal invasion of operation wound in, R.S.M. 68, 69
- Breast-feeding of infants, encouragement of, Odont. 6
- Breasts, ablation of, mutilation among female Skoptzi limited to, Path. 118
- Breath, odour of acetone in, in recurrent or cyclic vomiting and starvation, Child. 79, 82, 87
- Breathing, see *Back-breathing*, *Belly-breathing*, *Mouth-breathing*
- Breathing-exercises, Laryng. 42
- Breisky, kraurosis vulvæ, quoted, Obst. 29, 35
- BRIGGS, H.—Discussion on fibro-adenoma of ovary, Obst. 53
- Bright's disease, beneficial influence of climate of Egypt and North Africa on, Baln. 126
- , maladies classified under, Baln. 102
- Brill Hill, disused spa at, Baln. 41
- British Pharmaceutical Codex, deficiencies of, Therap. 92, 93, 94, 96, 100
- , statements as to contested, Therap. 100
- , reason for publication, Therap. 90
- , resolution of Pharmacopœia Committee of General Medical Council as to, Therap. 93, 97
- BROADBENT, Sir J. F. H., Bart.—Fibroid lung and displacement of heart, Clin. 133
- , discussion on pericarditis with effusion, Med. 77
- Brompton Hospital Staff, records of tuberculous infection, Med. 138-140
- Bronchiectasis, prognosis, Surg. 63
- , treatment by vaccines, R.S.M. 59, 114, 115
- Bronchitis, acute, treatment with vaccine, R.S.M. 67, 68
- , chronic, treatment with vaccine, R.S.M. 146
- , treatment by ether inhalation without admixture of exhalations from lungs (R. H. Hodgson), Clin. 213, 215
- Broncho-pneumonia (septic), result of pyorrhœa alveolaris, Odont. 29
- Bronchorrhœa, profuse, following intraspinal injection of magnesium-sulphate solution in treatment of tetanus, Med. 46
- Bronchus, soft foreign body in (D. R. Paterson), Laryng. 24
- , right, removal of mutton-bone from, by direct method (H. Tilley), Laryng. 127
- BRONNER, A.—Discussion on anatomical and pathological preparations, Otol. 26
- , discussion on early symptoms in case of epithelioma originating near right Eustachian tube, Otol. 31
- , on traumatic perforation through hard palate, Laryng. 146
- , on pedunculated papilliform growth, Otol. 46
- Brosius, cases illustrating effects of syphilitic virus on nervous system, quoted, Neur. 44
- Brother and sister, congenital enlargement of one limb occurring in (O. L. Addison), Child. 43
- BROWN, W. LANGDON.—Criticism of some principles in the treatment of chronic nephritis, Therap. 138-146; discussion (p. 146): Dr. J. Gray Duncanson, Dr. H. Charles Cameron, Dr. E. I. Spriggs, Dr. R. Paramore, Dr. Langdon Brown (reply)
- , discussion on treatment of gastric ulcer, Therap. 135
- , on therapeutic value of lactic-acid bacillus, Therap. 62
- and Gow, A. E.—Statistics of pericardial effusion from St. Bartholomew's Hospital, Med. 134
- Brown on pancreatic reaction, quoted, Med. 182
- Brown-Séquard's paralysis, see *Paralysis*, *Brown-Séquard's*
- , syndrome accompanying? syringomyelia (H. R. Prentice), Neur. 100
- BRUCE, J. MITCHELL.—Discussion on combination of auscultatory and tactile methods of reading arterial pressure, Med. 210
- , discussion on dorsal percussion of thorax and stomach, Med. 239

- BRUCE, J. MITCHELL.—Discussion on pancreatic reaction and faecal analysis, Med. 203
 —, discussion on rôle of fats in treatment of disorders of stomach, Med. 95
 —, on the treatment of tetanus, Med. 51
- BRUCE, W. IRONSIDE.—Discussion on pericardial effusion, Med. 93
- BRUCE-PORTER, H. E., MUMMERY, J. P. LOCKHART, and WHITE, W. HALE.—Case of pneumococcal colitis with hyperpyrexia, severe hæmorrhage treated by appendicostomy, Clin. 48; discussion (p. 51): Mr. Lockhart Mummery, Dr. Dalton, Mr. Charters Symonds, Dr. Hale White (reply)
- von Brünings' instrument for facilitating manipulations under direct laryngoscopy (G. S. Hett), Laryng. 3
- BRUNTON, Sir T. LAUDER.—Discussion on diagnosis and treatment of duodenal ulcer, Surg. 87
- Brush-discharges, bactericidal agents in, Electr. 84
- Buccal mucosa and circum-oral skin, pigmentation of, in pernicious anaemia, sequel to case (H. D. Rolleston), Clin. 216
- BUCHAN, G. F.—Discussion on scarlet fever: home treatment and prevention, Epid. 70
 —, discussion on summer diarrhoea and enteric fever, Epid. 269
- BUCHANAN, G. S.—Discussion on bovine tuberculous mastitis, Epid. 247
- BUCKLEY, C. W.—Discussion on arthritis deformans, Baln. 95
- BULLOCK, W.—Discussion on therapeutic value of lactic-acid bacillus, Therap. 58
 —, discussion on vaccine therapy, R.S.M. 75
- Bumm, on septic endometritis, quoted, Obst. 233
 —, results of rapid delivery in puerperal eclampsia, quoted, Obst. 201, 206, 207
- Bumm's clinic, results of myiotomy in, Obst. 124
- BUNCH, J. L.—Case of mycosis fungoides, Clin. 6; discussion (p. 8): Dr. de Havilland Hall
- Bungarus caeruleus* (common krait), venom of, action on central nervous system, Neur. 106, 109
 —, *fasciatus* (banded krait), venom of, action on central nervous system, Neur. 106, 108, 109, 112
- Burdet, rupture of tunica vaginalis in hydrocele, quoted, Surg. 163, 169
- BURGESS, W. MILNES.—Discussion on X-ray photographs from a boy the subject of habitual constipation, Child. 66
- BURGHARD, F. F.—Discussion on cases of epithelioma of larynx shown after operation, Clin. 119
- BURROWS, HAROLD.—Multiple exostoses, Clin. 164
 —, shortening of right humerus and left thigh due to unobserved fractures in early life (infantile scurvy?), Child. 165
 —, specimen of a chylous cyst of the mesentery associated with, and perhaps due to, enlarged glands in the mesentery; death from volvulus, Child. 164
- BUTCHER, W. DEANE.—Discussion on radium in treatment of malignant growths, Electr. 68
 —, discussion on treatment of nævi by electricity, cautery and refrigeration, Electr. 49
 —, on vaccine therapy [production of auto-vaccination by electrical methods], R.S.M. 153
 —, on X-ray treatment of lymphadenoma, Electr. 136
- Butler, E. A., "Our Household Insects," alluded to, Epid. 154
- BUTLER, G. G., and BOX, C. R.—Statistics of pericarditis with effusion from St. Thomas's Hospital, Med. 104
- BUTLER, W.—Discussion on control of scarlet fever, Epid. 86
- BUTLIN, H. T.—Definition of "leucoma" quoted, Laryng. 83
 —, diagnosis of malignant disease, quoted, Surg. 137
 —, discussion on treatment of inoperable sarcoma by mixed toxins, Surg. 45
 —, leucoplakic vulvitis, Obst. 29
- Butlin's method of operation for malignant disease of tongue, Clin. 184
- Buttocks and lumbar regions, blue patches on (F. Langmead), Child. 133
- Buxton, B. H., preparation of mixed toxins of *Bacillus prodigiosus* and *Streptococcus erysipelas*, Surg. 7
- BUXTON, DUDLEY W.—Discussion on heart massage in heart failure during anaesthesia, Anæsth. 10

- BUXTON, W. DUDLEY.—Discussion on lymphetism, *Anæsth.* 37
 —, the choice of the anæsthetic, an address introductory to a discussion on the subject. Discussion (p. 73): Mr. Gill, Dr. G. A. H. Barton, Dr. Silk, Dr. Llewelyn Powell, Dr. Dudley Buxton (reply)
- BUZZARD, E. FARQUHAR.—Case of tremor, *Neur.* 18; discussion (p. 19): Dr. Buzzard, Dr. J. Taylor
 —, discussion on case of acute cerebral softening, *Neur.* 132
 — of cerebellar sclerosis, *Neur.* 90
 — of spasmodic neurosis. ? *Paramyoclonus multiplex*, *Neur.* 29
 —, on distal type of myopathy, *Neur.* 94
 —, on lymphetism, *Anæsth.* 46
 —, olfactory discharges from organic disease, quoted, *Neur.* 2, 6
 —, secondary growths affecting spinal roots, with pathological report by C. M. Hinds Howell, *Neur.* 124, 125; discussion (p. 129): Dr. J. A. Ormerod, Dr. F. E. Batten, Dr. Howell (reply)
- BUZZARD, THOMAS.—Discussion on case of spasmodic neurosis. ? *Paramyoclonus multiplex*, *Neur.* 29
- BYTHELL, W. J. S.—Discussion on relative value of various transformers, *Electr.* 158
- Cachexia strumipriva, diminished intra-cartilaginous growth of bones, *Path.* 106
- Cæcum, acutely inflamed, appendix invaginated into lumen of, and forming apex of intussusception; operation; recovery (I. Back), *Child.* 25
 — tuberculosis of, and hypersecretion, *Surg.* 185
- Cæsarean section, abdominal compared with vaginal, *Obst.* 176
 — compared with Braxton Hicks' method in placenta prævia, *Obst.* 171-173
 —, extra-peritoneal, operation described, *Obst.* 119
 —, indications for, given at meeting of American Gynæcological Society, *Obst.* 174
 — in treatment of placenta prævia, case detailed (J. M. Munro Kerr), *Obst.* 11
 —, justifiability discussed, *Obst.* 13-15
 — of puerperal eclampsia, *Obst.* 205, 206 (F. J. McCann), *Obst.* 193-202
 — arguments against, *Obst.* 203
 —, effect on eclamptic fits, *Obst.* 200
 —, foetal mortality, *Obst.* 196, 198
 —, historical account of, *Obst.* 196, 197
 —, indications for, *Obst.* 198, 199
 —, maternal mortality, *Obst.* 196, 198
 —, precautions during operation, *Obst.* 199
 —, reservation for exceptional cases, *Obst.* 204, 205
 —, septic infection in, *Obst.* 199
 —, successful case of (F. J. McCann), *Obst.* 194
 — method of delivery in third and fourth degrees of contracted pelvis, *Obst.* 115
 —, place of, in the treatment of placenta prævia (H. Jellett), *Obst.* 167
 —, posterior, followed by total hysterectomy, for fibroids (H. R. Spencer), *Obst.* 62
 —, vaginal, *Obst.* 204, 207
- Cairo, see *Kasr-el-Ainy Hospital*
- Calcium, salts of, stimulate thyroid activity, *Baln.* 9
 —, and promote diuresis, *Baln.* 9
 — oxalate, cannot exist in blood, *Med.* 5
 —, derivation from oxalates of food-stuffs, *Med.* 4
 — found in renal tubules after experimental poisoning by oxalic acid, *Med.* 6, 7
 — in urine, in excess, *Med.* 1, 2; see also *Oxaluria*
 —, precipitation of, *Med.* 2, 3
 —, prevented by acid phosphate of sodium, *Med.* 2, 3

- Calcium oxalate in vegetables, Med. 4
 ———, insolubility of, Med. 2
 ——— crystals, presence in urine in chronic pancreatitis, Med. 168, 176
 ——— see also *Calculus (calcium oxalate)*
 Calculi contained in Meckel's diverticulum and producing colic (J. Sherren), Clin. 11
 Calculus, diagnosis by screen examination not wholly reliable, Electr. 96
 ———, disappearance of, in Buckingham Chilterns, Baln. 45
 ——— (calcium oxalate), diagnosis of, Med. 9
 ——— ———, hæmaturia due to, Med. 8, 9
 ——— ———, nature of, Med. 7
 ——— ———, solution of, by acid phosphate of sodium in urinary passages, Med. 11-13, 21
 ——— ——— ——— ——— ——— by laboratory experiment, Med. 13-20
 ——— (pancreatic), pancreatic reaction in subjects of, Med. 176
 ——— (renal), frequency compared with that of ureteral calculus, Electr. 94
 ——— ———, presence in both kidneys without symptoms, Electr. 91
 ——— ——— (or ureteral), position determined by aid of Röntgen rays, Electr. 88
 ——— (ureteral), diagnosis, by aid of Röntgen rays, Electr. 88
 ——— ———, diagnosis from gland shadows and phleboliths, Electr. 94
 ——— ———, points in (C. Thurstan Holland), Electr. 87-97
 ——— ———, frequency compared with that of renal calculus, Electr. 94
 ——— ——— (large), impaction of, in lower end of right ureter (A. Carless), Clin. 63
 ——— ———, methods of removal, Clin. 63, 64, 65
 ——— ———, tuberculous ulcer mistaken for, Electr. 91, 92
 ——— (urinary), determination of presence by radiography important, Med. 22, 23
 ——— ——— (renal and ureteral), bacterial causes to be sought for in connexion with, R.S.M. 16, 17
 ——— (vesical), radiograph produced through Snook's high-tension transformer, Electr. 158
 ——— ———, weighing eight ounces, removal of (A. Pearce Gould), Clin. 31
 Caldwell-Luc operation, Laryng. 57
 Calomel, intra-muscular injections of, in treatment of syphilis, Therap. 212, 235
 ——— ——— ———, benefit in severe forms, Surg. 212, 226
 ——— ——— ———, objections against and dangers from, Surg. 213
 CALVERT, J.—Discussion on teaching of therapeutics in hospital wards, Therap. 10
 CAMERON, H. CHARLES.—Discussion on criticism of some principles in treatment of chronic nephritis, Therap. 146
 CAMMIDGE, P. J.—Consideration of the results of the pancreatic (Cammidge) reaction in 1,475 cases, Med. 163; for discussion see below: *Cammidge (P. J.)*, diagnostic value of an analysis in the fæces, &c.
 ———, diagnostic value of an analysis of the fæces in diseases of the pancreas, Med. 185-202; discussion (p. 203): Dr. Mitchell Bruce, Dr. A. F. Hertz, Dr. H. D. Rolleston, Dr. H. D. McCulloch, Dr. Cammidge (reply)
 ——— and SEMON, H. C. G.—An experimental investigation into the origin and cause of the pancreatic (Cammidge) reaction in the urine, Path. 79-92
 CAMPBELL, HARRY.—Affection of posterior roots of lower cervical and upper dorsal nerves? tabes? lateral sclerosis, Neur. 34
 ———, discussion on cases of thrombosis of cerebral arteries and case of cerebral hæmorrhage from luetic vessels, Neur. 32
 ———, discussion on model demonstrating "back" and "belly" breathing, Laryng. 48
 ———, functions of thyroid gland, quoted, Baln. 8
 ———, on blood-plasma, quoted, Baln. 7
 Cancer, ætiology, general conception, Therap. 70
 ———, bacterial infection in, R.S.M. 17, 18
 ——— of breast, physiological capacity of cells, Path. 162
 ———, failure of organo-therapy in, Therap. 70, 71
 ———, immunity against degree of, Therap. 77

- Cancer, immunity against, results when established, Therap. 77
- reaction to (E. F. Bashford), Therap. 69-81
 - , signs of, Therap. 77
 - , immunization against plasma-cell reaction of connective tissue during process, Therap. 77
 - , reactions difficult or impossible to obtain, Therap. 77
 - , implantation of, immunity to, not a vaccination, Therap. 79
 - , mice protected against, developing tumours of own species, Therap. 79
 - , prevention, Therap. 80
 - limited to and ubiquitous among vertebrates, Laryng. 159, Therap. 70
 - , localization of development, Laryng. 159
 - , origin of *de novo* (autochthonal), Therap. 73
 - , prevalence of, in the Chilterns, Baln. 45
 - , protective inoculation of skin against, Therap. 76
 - , question of date of cure, illustrated by larynx of man, aged 78, whose right vocal cord was removed for epithelioma in September, 1896, with fresh epitheliomatous out-break on left cord (H. Tilley), Laryng. 33
 - , resistance to implantation of, compatible with suitability for implantation of sarcoma, Therap. 76
 - , squamous-celled, excision of larynx, lower part of pharynx, and upper end of œsophagus for (A. Evans), Clin. 44
 - , systematic experimental study, of but recent date, Therap. 80
 - , tissue-inoculation against, homologous or autologous, equally protective, Therap. 76, 77
 - , transference from one species to another impossible, Therap. 71, 72, 75
 - , transplantation of, Therap. 73, 75
 - , vaccine therapy of, R.S.M. 18
 - , not scientifically justifiable, Therap. 78, 79
 - , varieties and sub-varieties under observation, Therap. 74
 - of bladder, resection for (J. W. Thomson-Walker), Clin. 68
 - of breast, treatment by radium, case of, further report (N. S. Finzi and E. H. Shaw), Clin. 1
 - (endolaryngeal), case of, Laryng. 168
 - of larynx, extrinsic (G. S. Hett), Laryng. 53
 - , treatment by lateral pharyngotomy with partial laryngectomy, Laryng. 53, 54
 - of lung, diagnosis, Surg. 67
 - of nasopharynx, early symptoms, Otol. 29, 30
 - of ovary, engrafted on pre-existing cyst-adenoma, Obst. 97
 - , secondary, incidence compared with cancer of other organs, Obst. 97
 - of pharyngeal party-wall (W. Hill), Laryng. 142
 - of pylorus, treatment by radical operation (E. W. Hey Groves), Surg. 117-158
 - , squamous-celled, of dermoid cyst of ovary (E. H. Shaw), Obst. 143
 - of stomach affecting cardiac orifice, case of, with jaundice and areas of unpigmented skin (H. D. Rolleston), Clin. 195
 - , fats in treatment of, Med. 32, 34
 - , treatment, operative, reason for failures, Surg. 118
 - of uterus, course sometimes painless throughout, Obst. 5
 - of body of uterus, with keratinization of the growth (J. H. Targett), Obst. 153
 - , see also *Carcinoma; Serum (anti-cancer)*
- Cancer-cells, delicate nutritional requirements of, Surg. 11
- , destruction by toxins, Surg. 11
 - , great omentum carrier of, to pelvic organs, Surg. 148, 150
 - , implantation and continued growth of, reactions inimical to, how induced in living animals, Therap. 76
 - , paralysis of chemiotactic powers of, Therap. 77
 - , protein substances of, consequences following inoculation of, Therap. 77

- Canine teeth, immunity to caries, *Odont.* 80, 83
 — tooth and mandible vertically fractured (W. de C. Prideaux), *Odont.* 92
 Canities, case of (A. W. Williams), *Derm.* 110
 CANNEY, H. E. LEIGH.—Climate of Egypt and North Africa and its influence on disease, *Baln.* 115
 Cannon and Murphy, on pyloric spasm, quoted, *Surg.* 73
 Carbohydrates in intestinal tract, abnormal fermentation, *Therap.* 55, 56
 Carbolic oil, inunction with, in scarlet fever, *Epid.* 89
 —, treatment of scarlet-fever throat, *Epid.* 57
 Carbon-dioxide crayon, action of, *Electr.* 42
 —, dosage, *Electr.* 43
 —, preparation of, *Electr.* 44
 —, results of, *Electr.* 45
 —, in treatment of lupus erythematosus and vulgaris, *Electr.* 44
 — of moles and warts, *Electr.* 44
 — snow, raised vascular naevus cured by (J. M. H. MacLeod), *Derm.* 156
 Carbonates, mixed, administration of, in diagnosis of duodenal ulcer, *Surg.* 88, 89
 Carcinoma, antitryptic power of serum in, *Path.* 171
 —, "cell-nests," epithelial pearls, *Path.* 152
 —, commencing, and kraurosis of vulva (A. E. Giles), *Obst.* 54
 —, inoperable, extra-laryngeal, cases showing beneficial effects of operation on thyroid gland (W. Stuart-Low), *Laryng.* 13
 —, treatment with *Streptococcus erysipelas*, *Surg.* 4-6
 —, physiological capacities of cells, *Path.* 162
 —, primary, of Fallopian tube associated with acute inflammatory mischief (W. W. H. Tate), *Obst.* 75
 —, of liver, *Path.* 150
 —, recurrence after operations for, mixed toxins as prophylactic against, *Surg.* 16
 — of intestines, Röntgen diagnosis, *Electr.* 116
 — of œsophagus, case (W. Hill), *Laryng.* 120
 —, skiagrams, *Laryng.* 120
 — of ovary, specimen from (L. G. Anderson), *Obst.* 72 : discussion (p. 72) : Dr. Macnaughton-Jones
 — of pylorus, hypersecretion in, *Surg.* 186
 — of stomach, Röntgen diagnosis of, *Elect.* 115
 —, of vulva, leucoplakic vulvitis in its relation to (C. Berkeley and V. Bonney), *Obst.* 29
 —, kraurosis unknown in, *Obst.* 38
 —, pathology, *Obst.* 39
 —, treatment by radium, *Electr.* 64
 —, see also *Cancer*
 Carcinomata, multiple, of vulva, *Obst.* 45
 Carcinomatous tumour of arytaenoid, excision of half the larynx for (A. Evans), *Laryng.* 28
 Cardiac dullness, *Med.* 225
 —, diagnostic applications, *Med.* 225
 Cardiogram, character of, altered by intervention of pericardial fluid, *Med.* 101
 Cardiolysis in case of multiple serositis of tuberculous origin (F. J. Poynton and W. Trotter), *Clin.* 199, 200
 Cardiospasm in duodenal ulcer, *Surg.* 81
 Caries alveolaris specifica, *Odont.* 29
 —, dental, ætiological factors, exciting, *Obst.* 78
 —, predisposing, *Odont.* 78
 —, age-incidence, *Odont.* 82
 —, colour of teeth in relation to resistance to, *Odont.* 80, 82
 —, enamel sole barrier against acids causing, *Odont.* 73
 —, experimental production, *Odont.* 74

- Caries, dental, fermentation of food-stuffs and, *Odont.* 5
 ———, immunity of canines to, *Odont.* 80, 83
 ———, incapacitating effects of, *Odont.* 6
 ———, prevention by friction of teeth, natural and artificial, *Odont.* 82
 ———, progress rapid during typhoid and other fevers of long duration, *Med.* 77
 ———, ravages of, *Odont.* 6
 ———, relative susceptibility of different teeth to, experiments on (Stanley P. Mummery), *Odont.* 71
 ———, resistivity of enamel to, *Odont.* 81, 83
 ———, susceptibility to, inherited, *Odont.* 79
 ———, of teeth to, *Obst.* 78, 79
 CARLESS, A.—Discussion on case of (?) hyperplastic tuberculosis of pelvic colon, *Clin.* 206
 ———, discussion on cholecyst-duodenostomy for acute emaciation following formation of biliary fistula, *Clin.* 85
 ———, on Meckel's diverticulum containing calculi, *Clin.* 14
 ———, on surgery of malignant disease of the tongue, *Clin.* 187
 ———, on treatment of stricture of rectum and other conditions by fibrolysin, *Clin.* 89
 ———, excision of both maxillæ, *Clin.* 10; discussion (p. 11): Mr. Pearce Gould, Mr. W. G. Spencer
 ———, of both superior maxillæ: extension to frontal sinus, *Clin.* 114
 ———, impaction of large calculus in the lower end of the right ureter, *Clin.* 63; discussion (p. 64): Mr. Thomson-Walker, Mr. A. Pearce Gould, Mr. Carless (reply)
 ———, report on intestinal concretion found in a Meckel's diverticulum, *Clin.* 30
 ———, sarcoma of the ilium treated by Coley's fluid, *Clin.* 113
 CARLING, E. ROCK.—Lymphangioma of the tongue, *Clin.* 111; discussion (p. 111): Mr. E. Rock Carling, Dr. Parkes Weber, Mr. A. E. Barker
 CARLING, W.—Pseudo-hypertrophic muscular paralysis, *Child.* 158
 Carlsbad-water treatment of chronic gastric ulcer, *Therap.* 130
 CARLYLL, HILDRED B.—Enormous sarcomatous tumour in a child aged 2 years, *Child.* 154; discussion (p. 156): Dr. Carr, Mr. Mummery, Dr. Carlyll (reply)
 CARMALT-JONES, D. W.—Discussion on the bacteriology of pyorrhœa alveolaris, *Odont.* 66
 ———, discussion on vaccine therapy, *R.S.M.* 145
 CARMICHAEL, E. SCOTT.—Tuberculosis of the tonsil, associated with tuberculous glands of the neck, *Child.* 27-36
 Carnot, production of chronic pancreatitis in dogs by establishing route for introduction of intestinal bacteria from duodenum into pancreatic duct, *Path.* 83, 90
 CARPENTER, GEORGE.—Compression of the trachea by an enlarged thymus, *Child.* 46; discussion (p. 47): Dr. J. D. Rolleston, Dr. J. Porter Parkinson, Dr. Thursfield
 ———, microscopical section from the liver of a case of icterus in an infant, *Child.* 48
 CARPENTER, G. D. H., and ROLLESTON, H. D.—Scleroderma with sclerodactyly, *Clin.* 32; discussion (p. 34): Dr. J. Galloway, Dr. Herringham, Dr. Savill, Dr. Rolleston (reply)
 CARR, J. WALTER.—Case of (?) myositis fibrosa, *Child.* 147; discussion (p. 148): Dr. E. Cautley, Mr. L. Mummery, Dr. Carr (reply)
 ———, case of pneumococcal infection in an infant, simulating generalized tuberculosis, *Child.* 14; discussion (p. 16): Dr. Sutherland, Dr. Carr (reply)
 ———, discussion on ateleiosis in a man, *Child.* 146
 ———, on congenital pulmonary stenosis, *Child.* 150
 ———, on enormous sarcomatous tumour, *Child.* 156
 ———, on malignant endocarditis of tricuspid valve, *Child.* 142
 ———, specimen of the urinary organs of a boy who died apparently from uræmia; diphtheritic membrane being found in the trachea after death, *Child.* 90; discussion (p. 91): Dr. Cautley, Dr. Higgs, Dr. Carr (reply)
 Carriev and Legrand, fibromyoma of tube, quoted, *Obst.* 239
 CARTER, R.—Discussion on use of citrated milk for wasting infants, *Child.* 124.
 Cartilages (cricoid and arytenoid), tuberculous perichondritis of (H. Barwell), *Laryng.* 109

- CARWARDINE, THOMAS.—Microscopic appearances in tubal pregnancy, *Obst.* 157-161
 Casation, areas of, a hindrance in vaccine therapy of tuberculosis, *R.S.M.* 148
 CASSIDY, M. A.—Discussion on the treatment of tetanus, *Med.* 53
 Castration, hypoplasia of accessory sexual organs following, *Path.* 119
 — in male Belgian hare, effects on long bones, pelvis, and skull, *Path.* 104, 105
 — practised among Russian sect (Skoptzi), *Path.* 103
 Casual labourers, ill-effects of alcohol on, *Epid.* 6, 7
 — — — ranks recruited by cycles of trade depression, *Epid.* 6
 — — —, uncertain conditions of livelihood, in towns and sea ports, *Epid.* 6
 Cat, Wallerian degeneration of regenerated nerve fibres in, *Path.* 12, 22, 23, 24
 Cataract (discoid, familial or "Coppock"), pedigrees of, *Child.* 69
 — — —, two cases of (N. B. Harman), *Child.* 68
 Catarrh, intestinal, inducing slow changes in pancreas and resulting in diabetes, *Med.* 171
 —, long-standing, removal of hypertrophied anterior lip of hiatus semilunaris for (J. Dundas Grant), *Laryng.* 131
 CATHCART, G. C.—Complete occlusion of left nostril by deflected triangular cartilage in a girl aged 7½ years, *Laryng.* 56; discussion (p. 56): Mr. Stuart-Low, Dr. Pegler, Dr. H. J. Davis, Dr. Scanes Spicer, Dr. Fitzgerald Powell, Dr. Lambert Lack, Dr. Dan McKenzie, Mr. Herbert Tilley, Dr. Dundas Grant, Dr. Cathcart (reply)
 —, hoarseness of one month's duration in a woman aged 60, *Laryng.* 31; discussion (p. 31): Dr. Dundas Grant, Dr. Watson Williams, Dr. Cathcart (reply)
 —, further notes on a case of hoarseness in a woman aged 60, *Laryng.* 56; discussion (p. 56): Dr. Watson Williams, Dr. Dundas Grant, Dr. Cathcart (reply)
 —, tuberculosis of larynx in girl aged 12, *Laryng.* 127; discussion (p. 127): Dr. Jobson Horne, Dr. F. Powell, Dr. S. Spicer, Mr. C. Fox, Mr. Horsford, Dr. Donelan, Dr. W. Hill, Dr. J. Dundas Grant, Dr. G. C. Cathcart (reply)
 Catlin, effects of abstinence from salt among North American Indians, quoted, *Baln.* 61
 Cattle, effect of ingestion of, or abstinence from, common salt, upon, *Baln.* 61, 62
 —, possible phases of scarlet-fever infection in, *Epid.* 77, 78, 91
 — (short-legged breed, "Dexter" variety), how produced discussed, *Path.* 107, 108
 —, see also *Mastitis*, bovine tuberculous
 CAUSH, D. E.—Spindles of von Ebner, *Odont.* 144-49; discussion (p. 149): Mr. Hern, Mr. D. P. Gabell, Mr. J. Lewin Payne, Mr. E. B. Dowsett, Mr. H. Lloyd Williams, Mr. Caush (reply)
 Caution, electricity and refrigeration, treatment of nævi by (E. R. Morton), *Electr.* 37
 CAUTLEY, E.—Discussion on case of congenital thoracic deformity, *Child.* 102
 — discussion on case of myelogenous leukemia in infant, *Child.* 94
 — — on congenital pulmonary stenosis, *Child.* 151
 — — on diagnosis and treatment of intussusception in children, *Child.* 173
 — — on malignant endocarditis of tricuspid valve, *Child.* 142
 — — on (?) myositis fibrosa, *Child.* 148
 — — on specimen of hypertrophic stenosis of pylorus, *Child.* 100
 — — on specimen of urinary organs with presence of diphtheritic membrane in trachea, *Child.* 90
 "Cell-nests," epithelial pearls, *Path.* 152
 Cells as guide to estimation of immunity, *R.S.M.* 74
 Cellulitis, female, treatment by electricity, *Electr.* 7
 — (orbital), treatment by vaccine, *R.S.M.* 62
 Central Midwives Board, work of, *Obst.* 222, 232, 233
 Cerebellum, abscess of, relation to middle ear, *Otol.* 23, 26
 —, effector cells of, *Path.* 65
 —, fibres entering, distribution of, *Path.* 65
 —, sclerosis, case of (G. Holmes), *Neur.* 89
 —, tumour of, case of (W. J. Maloney), *Neur.* 99
 — — —, diagnosed instead of tumour of pineal body, *Neur.* 73

- Cerebrosides, group of lipid substances, Neur. 48
- Cerebrospinal fluid, examination of, in diagnosis of syphilis, Surg. 234
- examined by Wassermann reaction in cases of idiocy, Neur. 118, 123
- , increase of proteid in, in general paralysis and tabes, Neur. 39
- , lymphocytosis of, in general paralysis and tabes, Neur. 40-42
- , in parasyphilitic and syphilitic meningitis, Neur. 39
- , in secondary syphilis, Med. 36, 37
- Cervix, Bossi's mechanical dilatation of, in treatment of puerperal eclampsia, Obst. 204, 205, 207
- , carcinoma of, in prolapsed uterus in patient aged 86, with contact carcinoma on one of the labia, treatment by vaginal hysterectomy (H. R. Andrews), Obst. 161
- , degenerating fibromyoma of (F. McCann), Obst. 147
- , fibroid of cervix and body of uterus (J. P. Hedley), Obst. 189
- , very large fibroid tumour, successful operation for (A. E. Giles), Obst. 184
- Chaussaignac, views on laboratory work, quoted, R.S.M. 78
- Chalfont St. Giles, medical history, Baln. 40
- Chalkiness of enamel, first stage of decay, Odont. 72, 74, 75, 83, 84
- CHAMBERS, HELEN.—Method for studying the action of blood-fluids and other substances on the leucocytes, Path. 73
- Champetier-de-Ribes bag decreases foetal, increases maternal mortality, Obst. 175
- , use of, Obst. 123
- CHAMPNEYS, Sir F. H., Bart.—Discussion on Cæsarean section in placenta prævia, Obst. 178
- , discussion on modern methods of delivery in contracted pelves, Obst. 121
- , on slighter forms of puerperal sepsis, Obst. 231
- , remarks on occasion of delivery of presidential address (pain associated with disease of genital organs), Obst. 10
- Chancre, excision of, in treatment of syphilis, Surg. 229, 234
- Chantemesse, serum therapy in typhoid fever, R.S.M. 82
- Chapin, Dr., Fiske Memorial Prize Essay on control of scarlet fever, Epid. 86
- CHAPMAN, C. W.—Discussion on case of unidactyly, Child. 42
- Charcot, identity of rheumatoid with osteo-arthritis, Baln. 86, 87
- CHEADLE, B. A.—Case of disappearing abdominal tumour (shown for Percy Sargent), Clin. 85
- CHEATLE, A. H.—Discussion on anatomical and pathological preparations, Otol. 27
- , discussion on case of endothelioma of temporal bone, Otol. 33
- , on deformity of both pinnae, Otol. 71
- , on exostosis of right external meatus, Otol. 77
- , thanks to, with respect to presentation to Professor Politzer, Otol. 18
- , twenty specimens of chronic middle-ear suppuration and its sequelæ, eighteen of the bones being of the infantile types and two cellular, Otol. 41; discussion (p. 43): Dr. Pritchard, Mr. A. L. Whitehead, Mr. Waggett, Mr. H. E. Jones, Mr. Cheatle (reply)
- Cheek, inside of, lower lip and tongue, syphilitic ulceration of (C. Nourse), Laryng. 68
- , lips, tongue, larynx, fauces, and nasal cavities affected by pemphigus vegetans (H. J. Davis), Laryng. 78
- Cheeks, mucosa of, pigmentation in pernicious anæmia (H. D. Rolleston), Clin. 9
- Chelsea Hospital for Women, statistics as to relative malignancy of ovarian tumours at, Obst. 96, 97
- Chemico-parasitical processes acting on enamel, Odont. 81
- Chemists manufacturing, indiscriminate recommendation of lactic-acid bacillus by, Therap. 51, 52
- Child, case of lichen planus in (H. Davis), Derm. 104
- , extra-capsular fracture of neck of thigh-bone in (A. R. Thompson), Child. 22
- , multiple peripheral neuritis in (A. E. Garrod), Child. 38
- , symmetrical trophic lesion of extremities in (syringomyelia of Morvan's type) (H. Morley Fletcher), Clin. 98

- Child, symmetrical trophic lesion of extremities in, rarity of condition among children, Child. 103
- CHILDE, C. P.—Discussion on diagnosis and treatment of duodenal ulcer, Surg. 113
- , discussion on idiopathic dilatation of colon, Child. 182
- , multilocular cystic hygroma of neck, Child. 157; discussion (p. 157): Mr. Douglas Drew
- , partial tarsectomy for tuberculous disease, Child. 158
- , successful operation for gastric cancer, with involvement of other organs, Surg. 145
- , subperiosteal resection of the shaft of the ulna for tuberculous osteomyelitis, Child. 158
- , the diagnosis and treatment of intussusception in children, Child. 165-173; discussion (pp. 173-178): Dr. Cautley, Mr. Douglas Drew, Mr. Duncan C. L. Fitzwilliams, Mr. C. A. S. Ridout, Mr. Lockhart Mummery, Mr. Harold Burrows, Dr. Pernet, Mr. W. J. Midelton, Dr. L. Maybury, Mr. Childe (reply)
- Children, administration of ether to, Anæsth. 65
- of ethyl chloride to, Anæsth. 65
- born with syphilitic rash probably immune to re-infection, Neur. 46
- , cardiac dilatation in rheumatic pericarditis of, Med. 78
- , complete occlusion of nostril by deflected triangular cartilage in, question of operation by removal of projecting cartilage, Laryng. 57, 58
- — — — —, by submucous resection of septum, Laryng. 56-58
- , hospitals for, infection of children and nurses by patients suffering from diarrhoea, Epid. 105, 106
- , ill-effects of excessive ingestion of salt on, Baln. 71
- , immunity to untoward effects of chloroform not enjoyed by, Anæsth. 64
- , infectious and contagious diseases in schools, code of rules for prevention of in, Epid. 45
- , instruction to mothers amongst poorest classes as to care of, Epid. 34
- , intussusception in, diagnosis (C. P. Childe), Child. 165-173
- — —, treatment (C. P. Childe), Child. 165-173
- , latent appendicular disease in, Surg. 202
- , malnutrition in benefited by lactic-acid administration, Therap. 54, 55
- , medium-aged, anæsthetization with chloroform under surgical operations, Anæsth. 65
- , pneumococcal pericarditis in, Med. 94, 95
- , of poorest classes, beneficial effect of country life on, Epid. 5
- — —, condition of, Epid. 11, 12
- — —, ill-effects of town life on, Epid. 5
- — — — —, from want of exercise, Epid. 5
- — — — —, spread of diarrhoea owing to unclean habits of, Epid. 128
- , of same family, irregularity of appearance of congenital syphilis in, Neur. 46
- , paralysis following acute anterior poliomyelitis in, treatment by faradaic current, Electr. 105
- , performance of Killian's operation on, Laryng. 57, 58
- , pericardial effusion in, Med. 94
- , subjects of adenoids treated with vaccines, R.S.M. 215
- , of syphilitics, results of examination under serum reaction, Neur. 120
- (tuberculous, anæmic), treatment with tuberculin, results good, R.S.M. 116
- , tumour of pineal body in, symptom-complex associated with, Neur. 74, 75
- , ward space for, in isolation hospitals, same as for adults, Epid. 84
- (working-class), enforcement of due care for, on part of mothers, Epid. 43
- (young), ether followed by chloroform, routine anæsthetic for, Anæsth. 65, 66
- — —, mortality during operation for circumcision under chloroform, Anæsth. 65
- , see also *Feeble-minded children*
- Children's Hospital, Paddington Green, procedure of infant feeding at, Child. 107
- Childhood, duodenal ulcer in, Surg. 92
- Chill as cause of duodenal ulcer, Surg. 87, 106

- Chill, risk of, in climate of Egypt and North Africa, Baln. 125
- Chiltern Hills and Dales, dew ponds, bad human water-supply, Baln. 32, 45
- , geology, Baln. 32
- , health returns of present day, Baln. 44-46
- , meteorology of, Baln. 34, 35
- , natural and medical aspects (S. D. Clippingdale), Baln. 29-48
- , opium habit in, Baln. 36, 37
- , prevalence of cancer in, Baln. 45
- , racial characteristics of inhabitants, Baln. 35
- , rarity of tuberculosis in, Baln. 46
- Chiltern District, Rothschild villages, Baln. 46
- Chimiotropism of degenerated myelin attracting nerve-fibres, Path. 26
- Chimpanzees, infection with diarrhoeal faecal discharges, Epid. 96, 97
- Chin, tuberculous ulceration of, treatment with proteus vaccine, R.S.M. 21
- Chinese eunuch, generative organs and pelvis of, Path. 103, 124-126
- Chinese-ink method for detection of *Spirocheta pallida*, Surg. 219, 220, 238
- Chittenden, reduced protein diet, quoted, Therap. 141
- Chlorhydræmia, production of, in body, Baln. 68, 70
- Chlorides, excess of, failure of excretory organs to deal with, Baln. 78
- , retention of in body, effect of, Baln. 68
- Chloroform, abrogation of reflexes under, impossible, Anæsth. 65
- , administration in old age not advisable, Anæsth. 66
- , preferred to ether in cases of lymphatism, Anæsth. 32, 59
- , in small doses in cases of lymphatism, Anæsth. 38
- , special pathological conditions requiring, Anæsth. 71
- , to subjects of renal inadequacy safer than ether, Anæsth. 69
- , anæsthetization of medium-aged children with, under surgical operations, Anæsth. 65
- , ether preferred to, in secondary operations, Obst. 9
- , following scopolamine and morphine, anæsthetic recommended for neurasthenics, Anæsth. 67
- , mortality of young children under, during operation for circumcision, Anæsth. 65
- , no immunity to untoward effects among children, Anæsth. 64
- , preceded by ether, routine anæsthetic for young children, Anæsth. 65, 66
- , sudden death under, possible relation of thymus-gland secretion to, Anæsth. 51
- Chloroma, case of (J. C. G. Ledingham), Child. 97
- , autopsy, Child. 96
- , blood condition, case of (W. E. Wynter), Child. 96
- , sequel to, Child. 96
- , pathology of, Child. 96, 97
- Chocolate cream massolettes, Therap. 54, 59
- Cholecyst-duodenostomy for acute emaciation following the formation of biliary fistula (J. D. Malcolm), Clin. 83
- Cholera, immunity to, presumed to be due to abstinence from salt, Baln. 61
- , use of hypertonic salt solutions in, Baln. 74
- Cholesteatoma, indications for operative interference, Otol. 68
- , two cases of unusual size extending into posterior fossa and causing obliteration of sigmoid sinus (G. Wilkinson), Otol. 82
- , or keratosis obturans of external auditory canal: specimen (H. Todd), Otol. 81
- , pathogeny of (V. H. W. Wingrave), Otol. 63
- , (encapsuled), Otol. 63
- , (granulomatous), Otol. 66
- Cholesterin, Neur. 48
- , contents of colic found in Meckel's diverticulum, Clin. 13, 15
- Chondromatous growths, attempts to produce by grafting of foetal bones (S. G. Shattock, C. G. Seligmann, and L. S. Dudgeon), Path. 127-140

- Chorea and infective endocarditis, Therap. 101
- Chorio-epithelioma, primary, in Fallopian tube, Obst. 236, 237
- Chromatic changes in ganglion cells after cobra-venom poisoning, Neur. 110, 111
- Chromatolysis, diffuse, in cells of cerebral cortex, medulla oblongata, and spinal cord in specimens from fatal case of electric shock, Path. 145
- , perinuclear, Path. 16, 17
- , of Betz cells, Path. 17
- Chrome-silver method of Golgi, in demonstration of anatomy of nervous system, Path. 3, 4
- Chylous cyst (large) of mesentery (S. G. Shattock), Path. 98
- , description of specimen (S. G. Shattock), Path. 99
- , associated with enlarged glands in mesentery (H. Burrows), Child. 164
- of neck, excision (E. O. Thurston), Path. 97
- Cicatrices, treatment by static wave current, Electr. 104
- Cicatricial substitute, development of, for vocal cord in case of epithelioma of right vocal cord in man aged 60, Laryng. 156
- Cigarette smoke, inhalation of, causing laryngeal vertigo, Laryng. 79, 80
- Circumcision, mortality of young children under chloroform during operation for, Anaesth. 65
- Circum-oral skin and buccal mucosa, pigmentation of, in pernicious anemia, sequel to case (H. D. Rolleston), Clin. 216
- Cirrrosis, alcoholic, disappearance of chronic ascites in, after paracentesis and laparotomy (F. Parkes Weber and E. Michels), Clin. 167
- of liver, see *Liver, cirrhosis of*
- Cirrhotic facies, Child. 41, 43
- Citrated milk, undiluted, in feeding of wasting infants (F. Langmead), Child. 104
- Civatte, pathology of acne, quoted, Path. 172
- CLARE, T. C., and PARSON, J. INGLIS.—Prolapsus uteri with severe cardiac disease operated on under local and general spinal anaesthesia, Obst. 104
- CLAREMONT, CLAUDE C.—Case of idiopathic dilatation of the colon, Child. 178; discussion (p. 182): Mr. Lockhart Mummery, Mr. Childe
- , case of pemphigus, Child. 162
- of purpura, Child. 163; discussion (p. 163): Dr. Pernet
- CLARK, CHARLES A.—A method of ascertaining the relative position of unerupted teeth by means of film radiographs, Odont. 87; discussion (p. 89): Mr. W. Hern, Mr. D. P. Gabell, Mr. Clark (reply)
- , a sterilizable film-holder, Odont. 85
- Clarke, R. H., and Horsley, Sir V., on fibres entering cerebellum, quoted, Path. 65
- CLARKE, S. A., HERTZ, A. F., and ROWLANDS, R. P.—Case of phlegmonous gastritis from hydrochloric-acid poisoning; vomiting of the complete mucous membrane of the pyloric half of the stomach, Clin. 209
- Clarke's column, Path. 66
- , cells of, changes in, Path. 72
- Claudication (intermittent) due to chronic arteritis obliterans in tobaccoist (F. Parkes Weber), Clin. 96
- CLELAND, J. B.—Note on eosinophile cells in the exudate from tick-bites on a horse, Path. 42
- "Clicking" noise, objective and rhythmical, in left ear associated with synchronous muscular contraction of certain muscles forming floor of mouth (H. Tilley), Clin. 93
- , see also *Tics (clicking)*
- sound, objective, in left ear (H. Tilley), Otol. 36
- Climate, effects on internal secretory glands, Baln. 5
- , humidity controls subjective effects and immediate results of, Baln. 3, 4
- , influence on race, Baln. 2, 3
- , relation to cosmic influences, Baln. 12
- of Egypt and North Africa; its influence on disease (H. E. L. Canney), Baln. 115
- Climatology, future of, Baln. 12
- "Clinical" instruments, definition of, R.S.M. 76, 77

- Clinical medicine, importance of, to electro-therapeutists, Electr. 146
- Clinicians, attitude of, towards the scientific method, R.S.M. 75, 76, 77
- , ignorance of bacteriology, R.S.M. 9
- , indebtedness to bacteriologists, R.S.M. 76, 77
- , knowledge of bacteriology essential to, R.S.M. 113
- , limitations to vaccine therapy claimed by, R.S.M. 11, 12, 13, 108-112
- , position towards study of bacteriology, R.S.M. 4, 102, 111
- , relations with bacteriologists, R.S.M. 9, 60, 61, 110, 111, 117, 143, 145, 187, 210
- , subservency of laboratory workers to, R.S.M. 78
- CLIPPINGDALE, S. D.—The Chiltern hills and dales in certain of their natural and medical aspects, Baln. 29-48
- Clogg, H. S., diagnosis of intussusception in children, quoted, Child. 167
- , statistics of intussusception in children, quoted, Child. 170
- Clover's inhaler in administration of ether, when used with bag, dangerous, Clin. 215, 216
- Clutton, H. H., the late, tribute to the memory of (A. Pearce Gould), Clin. 29
- Cobra-venom, injection of, recovery after, Neur. 108
- , poisoning by, action on central nervous system, Neur. 106
- Cocaine, intraspinal (intrathecal) injection, effects on strychnine poisoning shown experimentally, Med. 52
- Coccygeal percussion, Med. 230
- Coccygodynia, pain of, Obst. 8
- Cockroaches, possible agency in spread of typhoid fever, Epid. 209-11
- Cohnheim, olive oil in treatment of gastric disorders, quoted, Med. 26, 27
- COLE-BAKER, L.—Absence of puncta lachrymalia (bilateral), Child. 159
- COLEBROOK, L.—Discussion on vaccine therapy, R.S.M. 41
- COLEMAN, F.—Exostoses of the mandible, Odont. 21; discussion (p. 24): Mr. Hern, Mr. C. Lucas, Mr. F. J. Bennett, Mr. Warwick James, Mr. H. Baldwin, Mr. Coleman (reply)
- , papillomata of the uvula, Odont. 20
- COLEY, W. B.—The treatment of inoperable sarcoma by bacterial toxins (the mixed toxins of the *Streptococcus erysipelas* and the *Bacillus prodigiosus*), Surg. 1-44; discussion (p. 44): Mr. Warrington Haward, Mr. Butlin, Sir A. E. Wright, Dr. Coley (reply)
- Coley's fluid in treatment of sarcoma of ilium, Clin. 113
- Colic and biliary hypersection, Surg. 184
- , appendicular, Surg. 206
- , produced by calculi contained in Meckel's diverticulum (J. Sherren), Clin. 11
- Colitis associated with adnexal affection, pain due to, Obst. 8
- , bacterial diagnosis of, use of vaccines in, R.S.M. 63, 64
- , benefited by lactic-bacillus administration, Therap. 54, 59, 60, 62
- , (mucous) benefited by lactic-bacillus administration, Therap. 54, 60
- , causal organism determined by auto-inoculation in combination with opsonic index, R.S.M. 46
- , (pneumococcal) with hyperpyrexia; hæmorrhage treated by appendicostomy (H. E. Bruce-Porter, J. P. Lockhart Mummery, and W. Hale White), Clin. 48
- , treatment by vaccine, R.S.M. 104, 105
- , (ulcerative), treatment by vaccine, R.S.M. 58
- Collargol in treatment of endocarditis, Therap. 105, 108, 112, 115, 116
- , cases illustrating, Therap. 105
- Colles's law in congenital syphilis, Neur. 45
- COLLINSON, HAROLD.—Analysis of 197 cases of duodenal ulcer operated upon by Mr. Moynihan (1900-1908), Surg. 79
- Colman, W. S., hallucinations produced by organic disease, quoted, Neur. 9
- , special sense discharges from organic disease, quoted, Neur. 2, 4, 9
- Colon, cleansing of, by injection of lactic-acid bacillus preliminary to operation, Clin. 203
- , (dilatation, idiopathic), fatal case of (C. C. Claremont), Child. 178
- , treatment by colotomy, Child. 180, 182

- Colon (dilatation, idiopathic), treatment, operative, discussed, Child. 181, 182
 —, hyperplastic growths of, diagnosis, Clin. 207
 —, pelvic, ? hyperplastic tuberculosis of, case of (J. P. Lockhart Mummery), Clin. 201
 —, —, —, operation for, Clin. 203
 —, —, —, specimen described, Clin. 204
 —, stricture of, non-malignant, Clin. 207
 Colostomy in intussusception, Child. 169
 COLYER, J. F.—Discussion on bacteria of pyorrhœa alveolaris, Odont. 65
 — discussion on case of painful attrition, Odont. 120
 — on treatment of dental cysts invading the antrum, Odont. 117
 — on unilateral overgrowth of lower jaw, Odont. 49
 —, report as Hon. Curator of Museum (Odontological Section), Odont. 143
 — and Smale, on absorption of roots of permanent teeth, quoted, Odont. 14
 "Comforters," alleged conveyance of diarrhœal infection by, Epid. 124
 Common lodging-houses, selection of inmates, Epid. 37
 —, spread of diseases in, Epid. 37
 — in Manchester, Epid. 26
 —, focus of infection for phthisis, Epid. 27
 Complement, deviation of, R.S.M. 163
 —, discovery, R.S.M. 4
 —, bodies producing, R.S.M. 92
 Complement-fixing-power of serum of syphilitics, Surg. 239
 Complement-fixing-test in diagnosis of syphilis, Surg. 227, 239
 Conjunctiva, tuberculosis of, treatment by vaccine inoculations, R.S.M. 61
 Conjunctivitis, treatment by vaccine inoculations, R.S.M. 62
 Constipation (chronic), treatment by static wave current with rectal electrode, Electr. 102
 —, different forms, diagnosed by Röntgen rays, Electr. 118
 —, fats in treatment of, Med. 33, 34
 — (habitual), X-ray photographs from boy aged 3, the subject of (E. I. Spriggs and H. F. L. Hugo), Child. 64
 — in relation to condition of teeth, Odont. 102
 — (obstinate), treatment by recto-abdominal application of static electricity, Electr. 82
 —, selected cases benefited by lactic-acid administration, Therap. 55
 — in women, treatment by electricity, Electr. 7, 14, 15, 18
 Consultants and general practitioners, responsibility for correct diagnosis and treatment of cases falls jointly upon, R.S.M. 6
 Consultation, cases for, compared with laboratory reports on specimens, R.S.M. 6, 7
 — by correspondence, deprecation of, R.S.M. 8, 9
 Contagious diseases in schools, code of rules for prevention, Epid. 45, 46
 Contagious Diseases Act, effect of, Surg. 247
 Contrexéville, waters, diuretic action explained, Baln. 9
 Controls in determination of value of vaccine therapy, R.S.M. 139, 140
 Convulsions, death under, Anæsth. 50
 —, epileptiform, in cerebral syphilis, Neur. 52, 62, 63
 —, recurring left-sided, with transient motor aphasia, in a left-handed woman (J. Taylor), Neur. 17
 Cooking, instruction in, Odont. 2
 COPENMAN, S. M.—Discussion on summer diarrhœa and enteric fever, Epid. 257
 —, earliest record of oral administration of vaccine (1810), quoted, R.S.M. 198
 "Coppock" cataract, see *Cataract, discoid familial*
 Cork maternity statistics (1872-77), Obst. 126, 127
 CORNER, EDRED M., and HUGGINS, MARTIN.—Pyæmic knee- and shoulder-joints treated by tapping and incision, Clin. 120; discussion (p. 121): Mr. A. E. Barker, Dr. Parkes Weber
 Correspondence, consultation by, deprecated, R.S.M. 8, 9

- Corvisart, advances in dorsal percussion made by, *Med.* 212
- Cosmic laws, influences of climate and, *Baln.* 12
- on human race, *Baln.* 1, 2
- on sexes, severally, *Baln.* 1, 2
- on types of mankind, *Baln.* 12
- Cough (brassy), clinical indication of diagnosis of aneurysm of descending thoracic aorta, *Clin.* 126
- Counter-irritation in treatment of rheumatoid arthritis, *Baln.* 109-111
- Country life, beneficial effects on children of, poorest classes, *Epid.* 5
- COUTTS, F. J. H.—Discussion on bovine tuberculous mastitis, *Epid.* 248
- Cow-sheds, inspection of, for bovine tuberculous mastitis, *Epid.* 220-225
- , state of, a factor in prevalence of bovine tuberculous mastitis, *Epid.* 231-239
- COWAN, J.—Treatment of endocarditis, *Therap.* 101; discussion (pp. 113-116): Dr. J. Mackenzie, Dr. Sainsbury, Dr. Morison, Dr. Pitt, Dr. Cowan (reply)
- Cows, age of, in relation to bovine tuberculosis, *Epid.* 235, 241
- , condition of herd, as factor in prevalence of bovine tuberculous mastitis, *Epid.* 231-239
- in state of advanced tuberculosis, chief factor in spreading disease, *Epid.* 239, 242, 255, 256
- , prevalence of bovine tuberculous mastitis among, *Epid.* 217-243
- CRACE-CALVEET, G. A.—Discussion on vaccine therapy, *R.S.M.* 158
- Craig, vaccine treatment of pneumonia, quoted, *R.S.M.* 103
- Craniotomy, justifiability of, questioned, *Obst.* 115, 116
- Cream, use of preservatives, in, *Child.* 105
- Creasote inhalation, *Therap.* 18, 19, 20
- Credé, introduction of collargol by, *Therap.* 105
- Cretinism, diminished intra-cartilaginous growth of bones in, *Path.* 106
- (fœtal), case alluded to, *Path.* 110
- Cricoid cartilage, relation to spinal column in back- and belly-breathing (R. H. Scanes Spicer), *Laryng.* 41
- , transverse axis of respiratory rotation of, on thyroid cartilage in belly-breathing, illustrated by model (R. H. Scanes Spicer), *Laryng.* 41, 44, 49
- Critien, Dr., diarrhoea in Malta, *Epid.* 104
- Crocker, H. Radcliffe, dietetic rules for patients suffering from skin diseases, *Baln.* 77
- Crooksbury Sanatorium, records of tuberculous infection of staff, *Med.* 139
- CROOKSHANK, F. G.—Discussion on scarlet fever: home treatment and prevention, *Epid.* 70
- , the control of scarlet fever, *Epid.* 73; discussion (p. 86): Dr. Butler, Dr. E. W. Goodall, Dr. Milne, Dr. Hamer, Dr. Niven, Dr. Crookshank (reply)
- Croom, Sir J. Halliday, cases of Cæsarean section in eclampsia, quoted, *Obst.* 197
- Cross-infection in isolation hospitals, *Epid.* 79, 87, 94
- Cryptorchism, bilateral, *Path.* 52, 53, 54
- Cubicle system, arrangement of cases in isolation hospitals by, *Epid.* 82, 83, 89
- Curette, use of in septic endometritis, reasons against, *Obst.* 233
- Curling, T. B., bilateral cryptorchism, quoted, *Path.* 52, 53, 54, 55
- CURRIE, A. S.—Discussion on lymphatism, *Anæsth.* 60
- CUSHNY, A. R., F.R.S.—Discussion on action of some diuretics, *Therap.* 50
- , discussion on proprietary, patent, and secret remedies, *Therap.* 99
- on teaching of therapeutics in hospital wards, *Therap.* 15
- on treatment of incipient pulmonary tuberculosis by antiseptic inhalations, *Therap.* 37
- Cuticle, extension of, inside nostrils (J. Donelan), *Laryng.* 90
- Cutter, advocacy of electric treatment in gynecology, *Obst.* 27
- Cyanosis, case of, with venous obstruction (W. P. Herringham), *Clin.* 168
- , choice of anæsthetic for subjects of, *Anæsth.* 70
- Cynotoxin (apocynamarin), *Therap.* 46
- Cyst of humerus, diagnosis from sarcoma, *Child.* 99

- Cyst of humerus, fracture, operation (H. Lett), Child. 98
 — (innocent), diagnosis from sarcoma of bone, Clin. 195
 — —, fracture of humerus at site of (R. C. Elmslie), Clin. 192
 — (teratomatous), secondary implantation (V. Bonney), Obst. 151
 —, see also *Chylous cyst*
 Cystectomy, see *Bladder, excision of*
 Cystic degeneration of breasts and ovaries, supposed toxic origin, R.S.M. 13, 14
 — fluid, origin of, Odont. 125-127
 — swelling at root of nose (P. M. Heath), Child. 71
 Cystitis, symptoms, as guide to progress and treatment of case, R.S.M. 125
 — (acute), treatment by vaccine, R.S.M. 184, 185
 — in women, treatment by electricity, Electr. 7, 10, 12, 13, 15, 18, 29
 Cystoscopy, importance of, in diagnosis of diseases of bladder, Clin. 74
 Cysts, classification varied in different editions of "Nomenclature of Diseases," Odont. 161
 — (dental), etiology and pathology of, Odont. 123
 — —, origin of cystic fluid, Odont. 125-127
 — —, suppurating, invading maxillary antrum; method of treatment (H. Tilley), Odont. 116
 — — — — —, two cases (H. Tilley), Odont. 115
 — (dentigerous), Odont. 159, 160
 — —, fibrous tissue surrounding, Odont. 159, 160
 — —, fluid in, Odont. 159
 — —, loose use of term, Odont. 161
 — —, teeth in, nature of, Odont. 159
 — of jaws, Odont. 121-142, 158
 — —, origin of cystic fluid, Odont. 125-127
 — —, various species, Odont. 123
 —, pancreatic, pancreatic reaction in cases of, Med. 177
- Daboia russellii* (chain viper), venom of, action on nervous system slight, Neur. 107
 Dachsund, histological details of ossification of long bones in, Path. 108-110
 —, short limbs of, how produced, discussed, Path. 107, 108
 —, thyroid administration to, with negative result, Path. 108
 Da Costa, J. C., jun., practical value of spinal percussion for the diagnosis of diseases of the mediastinum, Med. 215
 DALE, H. H., and LAIDLAW, P. P.—Action of some diuretics, Therap. 38; discussion (p. 50): Prof. A. R. Cushny
 DALLY, J. F. HALLS.—Discussion on model demonstrating "back" and "belly" breathing Laryng. 49, 50
 DALTON, N.—Discussion on pneumococcal colitis with hyperpyrexia; hæmorrhage treated by appendicostomy, Clin. 51
 — discussion on thoracic aneurysm in boy, Clin. 37
 DANIEL, P.—Discussion on condition of mouth in chronic disease, Odont. 108
 Darier's disease, case of (A. W. Williams), Derm. 95
 Dark-ground condenser for detection of *Spirochaeta pallida*, Surg. 219
 Daughter and mother, congenital oedema with cardiac defect in (F. J. Poynton), Child. 66
 DAVIES, Lieut.-Col. A. M.—Discussion on summer diarrhoea and enteric fever, Epid. 262
 DAVIES, SIDNEY.—Discussion on communication of diarrhoea from sick to healthy, Epid. 125
 DAVIS, E.—Discussion on removal of penny from gullet, Laryng. 136
 DAVIS, HALDIN.—Case for diagnosis, Derm. 105
 —, case of granuloma annulare, Derm. 57
 — — of lichen planus in a child, Derm. 104
 —, linear naevus of unusual type (naevus acneiformis), Derm. 68
 —, sections from a case of linear naevus, Derm. 105

- DAVIS, H. J.—Adventitious membrane, resembling normal drum, formed after radical mastoid operation, Otol. 58; discussion (p. 59): Mr. H. Tod, Dr. F. Powell, Dr. Kelson, Mr. H. E. Jones, Dr. Milligan, Dr. Davis (reply)
- , case of acute pemphigus of larynx in woman aged 40, Laryng. 22; discussion (p. 22): Mr. Baber, Dr. Paterson, Dr. Davis (reply)
- , case for diagnosis, Laryng. 139; discussion (p. 140): Mr. A. Thorne, Dr. Dundas Grant, Dr. Davis (reply)
- , case of excision of the entire tongue, with its results, Laryng. 137; discussion (p. 138): Dr. Dundas Grant, Mr. D. Armour, Mr. C. Nourse, Mr. A. Evans, Dr. D. McKenzie, Dr. Davis (reply)
- , case of laryngeal vertigo in a man aged 47, Laryng. 79; discussion (p. 79): Dr. Watson Williams, Dr. de Havilland Hall, Dr. Pegler, Dr. FitzGerald Powell, Dr. Scholefield, Dr. Dundas Grant, Dr. Watson Williams, Dr. Davis (reply)
- , deformity of both pinnae resulting from perichondritis following double mastoid operation, Otol. 71; discussion (p. 71): Mr. A. Cheate, Dr. W. Milligan, Mr. West, Mr. H. Tod, Dr. Dundas Grant, Dr. Davis (reply)
- , demonstration of Hay's pharyngoscope, Laryng. 23
- , discussion on bands between Eustachian tube and pharynx, Laryng. 70
- , on case of asthma; improvement after nasal treatment, Laryng. 117
- , of audible tinnitus, Otol. 1, 54
- , of chronic laryngitis with ? prolapse of ventricle on right side, Laryng. 30
- , of congenital occlusion of left posterior nares, Laryng. 92
- , of laryngeal growth, Laryng. 39
- , of paresis of palate, pharynx, and œsophagus, Laryng. 91
- , of probable malignant disease of larynx in a woman, Laryng. 37
- , on early symptoms in case of epithelioma originating near right Eustachian tube, Otol. 30
- , on Hay's pharyngoscope, Otol. 10
- , on model demonstrating "back" and "belly" breathing, Laryng. 48, 50
- , on new growth of right tonsil, Laryng. 148
- , on osteomyelitis of right temporal bone, secondary to mastoid disease, Otol. 58
- , on party-wall pharyngeal cancer, Laryng. 143
- , on peculiar erythema of left palate in a smoker, Laryng. 27
- , on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 57
- , specimen of larynx showing re-development of epithelioma on left cord after removal of right for same cause (September, 1896), Laryng. 34
- , on two cases of hoarseness, Laryng. 40
- , fracture of hyoid bone in a man aged 56 ("Garrotter's throat"), Laryng. 77
- , fungating mass on the epiglottis, with implication of the larynx, in a man aged 45, Laryng. 140
- , large tuberculous ulcer on the right side of the tongue in a man, Laryng. 81; discussion (p. 82): Dr. Watson Williams, Dr. Dundas Grant, Dr. Davis (reply)
- , laryngeal case for diagnosis: abductor paralysis right side, abductor paresis left side, Laryng. 119
- , left abductor paralysis in a man aged 64, case for diagnosis, Laryng. 136; discussion (p. 137): Dr. Dundas Grant, Mr. Barwell
- , facial paralysis following a mild catarrh of the middle ear; no perforation, Otol. 60, discussion (p. 60), Dr. D. Grant, Dr. Davis (reply)
- , ? papilloma on the posterior superior quadrant of the right drum, Otol. 60
- , pemphigus vegetans affecting the larynx and fauces, nasal cavities, tongue, cheek and lips of a woman aged 40, Laryng. 78; discussion (p. 78): Dr. StClair Thomson, Dr. W. Hill, Dr. Dundas Grant, Dr. Davis (reply)
- , perforation of nasal septum from salt dust, Laryng. 5
- , two cases of foreign body (a) in the lung, (b) in the œsophagus, Laryng. 75; discussion (p. 76): Dr. W. Hill, Mr. Tilley, Dr. Davis (reply)

- Dawbarn's method of operation for malignant disease of tongue, Clin. 184
- Dawson, B., hæmorrhagic gastralgia, quoted, Surg. 205
- DAWSON, G. W.—Bazin's disease in a man aged 36, Derm. 27
- , case of dermatitis herpetiformis, Derm. 26
- , — for diagnosis, Derm. 27; discussion (p. 27): Dr. Pringle, Dr. Pernet
- , — of pruritus, Derm. 16; discussion (p. 16): Dr. Savill
- , — of rodent ulcer, Derm. 47
- , dermatitis herpetiformis in a man aged 58, Derm. 15
- , discussion on case of chronic tuberculosis cutis, Derm. 54
- , myxœdema in a woman aged 41, Derm. 84
- , two cases of urticaria, Derm. 47; discussion (p. 47): Dr. Stowers
- Deaf, persons partially, acoustic aid for (H. Macnaughton-Jones), Otol. 40
- Deaf-mutes, ear of, pathological changes found in (lantern-slide demonstration) (A. A. Gray), Otol. 11
- Deafness, hysterical, with active vestibular reactions (P. H. Abercrombie and D. McKenzie), Otol. 74
- , tabetic, case of (D. McKenzie), Otol. 38
- DEAN, H. R.—Examination of the blood-serum of idiots by the Wassermann reaction, Neur. 117; discussion (p. 122): Dr. Shuttleworth, Dr. Head, Dr. Ormerod, Dr. Dean (reply)
- Decidual reaction extending from endometrium to tubal mucosa cause of tubal pregnancy, Obst. 238
- Decker, on pancreatic reaction, quoted, Med. 181
- Deformity of both pinnae resulting from perichondritis following double mastoid operation (H. J. Davis), Otol. 71
- Deinbollea Nyikensis*, Baker (kangalugi root), Therap. 67
- Deiter's nucleus, Path. 66, 70
- , —, cells of, changes in, Path. 72
- , —, — degenerated fibres round, Path. 71
- , —, changes in, Path. 66, 68, 71
- DELÉPINE, A. SHERIDAN.—Contribution to the study of the influences determining the prevalence of bovine tuberculous mastitis, Epid. 217-43; discussion (p. 244): Dr. J. Niven, Dr. C. J. Martin, F.R.S., Sir Shirley Murphy, Mr. M. Greenwood, jun., Dr. M. Richards, Dr. G. S. Buchanan, Professor Simpson, Dr. Coutts, Dr. Delépine (reply)
- , summer diarrhoea, quoted, Epid. 132
- Delivery, by high forceps, when dangerous, Obst. 128
- , modern methods of, in contracted pelves (E. Hastings Tweedy), Obst. 113
- , normal, in first and second degrees of contracted pelvis, Obst. 114, 120
- , obesity complicating, Obst. 114, 122
- , rapid, in puerperal eclampsia, Obst. 200, 201, 202
- Delore, experiments on power of resistance of tunica vaginalis, quoted, Surg. 167, 168, 169
- Dementia paralytica juvenilis, ultimate result of congenital lues, which caused optic atrophy in (H. Head), Neur. 33
- Dendy, Miss M., establishment of homes for feeble-minded children, Epid. 41
- Denker's operation, extension of, in combined maxillary and ethmoidal operation for endothelioma, Laryng. 67
- Dental capsule, histology of, Odont. 127-132
- operation, rupture of gastric ulcer during (W. de C. Prideaux), Odont. 91
- surgeons, removal of odontocoeles by, Odont. 158
- Dentine, decalcification, rates of, Odont. 73
- Dentistry, bearing of, upon public health, Odont. 1-8
- , condition of, amongst ancient Egyptians, Odont. 155
- , relation to general surgery, Odont. 4
- , — to other branches of medicine, Odont. 1-8
- , vaccine therapy in, R.S.M. 62, 86

- Dercum's disease, see *Adiposis dolorosa*
- Dermatitis artefacta, case of (T. Colcott Fox), Derm. 28
- , in hysterical subjects, Derm. 28, 29
- , always accompanied by anaesthesia of soft palate, Derm. 29
- (chronic superficial in patches), two cases of, with symmetrical distribution (H. G. Adamson), Derm. 65
- herpetiformis, cases of (P. S. Abraham), Derm. 26
- (G. W. Dawson), Derm. 15, 26
- (J. E. R. McDonagh), Derm. 89
- , syphilitic, case of (J. T. Leon), Child. 159
- , "trophic," commencing on the extremities (J. Galloway), Clin. 139
- , X-ray, chronic, refrigeration in treatment of, Electr. 44
- Dervishes (skulls of), condition of teeth in, Odont. 154, 155
- Desert climate, features of, Baln. 116
- Detention homes for feeble-minded children, Epid. 41
- Dew ponds, bad human water supply in the Chilterns, Baln. 32, 45
- Diabetes, result of intestinal catarrh slowly inducing changes in pancreas, Med. 171
- , bacterial origin, R.S.M. 18
- (bronzed), with cirrhosis of liver and pancreas and pigmentary deposits in liver, pancreas and skin (H. French), Path. 94
- (chronic), association of lichen planus with, case of (J. Galloway and A. W. Williams), Derm. 45
- , treatment by vaccine, R.S.M. 18, 178-181
- Diabetin, Therap. 86
- Diagnosis, cases for (H. G. Adamson), Derm. 55
- (H. Davis), Derm. 105
- (H. J. Davis), Laryng. 139
- (E. G. Graham Little), Derm. 29, 70
- (J. M. H. MacLeod), Derm. 112
- (W. K. Wills), Derm. 12
- abductor paralysis right side, abductor paresis left side (H. J. Davis), Laryng. 119
- , left side (H. J. Davis), Laryng. 136
- laryngeal new growth (P. Watson Williams), Laryng. 60
- papilloma (large) of larynx (A. Thorne), Laryng. 92
- , xantho-erythrodermia perstans (G. W. Dawson), Derm. 27
- Diagnosis, difficulties in, how to solve, R.S.M. 110, 111
- , importance of pain in, Obst. 6, 7
- , value of opsonic index in, R.S.M. 45
- , vertebral percussion in, Med. 215
- (bacterial) of appendicitis, R.S.M. 68
- , value of opsonic index in, R.S.M. 94, 95, 106, 190
- of vaccine therapy in, R.S.M. 63, 64
- (physical), functional errors in, R.S.M. 6
- , importance of, R.S.M. 110, 111
- , use of laboratory tests cannot replace or dispense with, R.S.M. 111
- (surgical), vertebral percussion in, Med. 215
- Diaphoresis, in treatment of chronic nephritis, Therap. 146
- Diaphragm, movements of, Laryng. 46, 49, 50, 51
- , arrest and inhibition following pericarditis, Med. 82, 83
- , followed by massive collapse of left base of lung, Med. 83
- Diaphragmatic region, gravitation of pus or serum to, dangerous, Obst. 8, 9
- Diarrhoea, aetiology, deductions made, based on fatal cases among infants at Manchester, Epid. 103, 104
- , benefited by lactic-bacillus administration, Therap. 54
- , boiling of milk alleged to be no protection from, Epid. 125, 128

- Diarrhoea, case histories, conclusions from, Epid. 111
 —, cases of, in houses where deaths occurred, details, Epid. 108
 —, in houses where no deaths occurred, details, Epid. 109
 —, causative agent, no particular organism can be claimed as, Epid. 129
 —, communication of, from sick to healthy (J. E. Sandilands), Epid. 95-121
 —, arguments against, Epid. 123, 124
 —, capability proved by experiments on chimpanzees, Epid. 96, 97
 —, disbelieved or held in question, Epid. 107, 108
 —, conclusions derived from case histories, Epid. 107
 — (chronic), due to excessive ingestion of salt, Baln. 65, 71
 —, death-rates in Manchester, 1891-1906, Epid. 176, 177
 —, effect of water-carriage system of sewerage on, Epid. 111-118
 —, epidemic at Helmsley, Epid. 96
 —, faecal discharges intensely contagious, Epid. 96
 —, proved by infection during laboratory examination, Epid. 97
 —, incidence on those in contact with sick in hospitals, Epid. 105
 —, infants suffering from pneumonia attacked with, Epid. 107
 —, infection of nurses with, Epid. 130
 —, of patients in hospitals with, Epid. 106, 107, 108
 —, multiple cases in houses and families with no fatal cases, Epid. 110
 —, notification of, Epid. 125
 —, outbreak (isolated) at Winchester, Epid. 97, 98
 —, spread of, alleged to be by means of "comforters," Epid. 124
 —, by agency of flies, Epid. 127
 —, by flies in milk, Epid. 129
 —, by unclean habits of children of poorer classes, Epid. 128
 —, social condition of population a factor, Epid. 122, 128
 —, through milk, Epid. 125
 —, symptom of rupture of tunica vaginalis in hydrocele, Surg. 167
 —, transmission by flies, Epid. 97
 — in England, greatest prevalence in urban districts, Epid. 104
 — in Ipswich, diminution in prevalence, Epid. 112, 113, 117, 119
 — in Kensington, fortnightly incidence compared with that of whole of London, Epid. 100
 —, influence of aggregation on, Epid. 104
 —, multiple cases, consecutive dates of onset, Epid. 102, 103
 —, in houses where deaths occurred, Epid. 102
 —, invasion of houses and families, date of onset of cases, Epid. 100, 101
 — in Leicester, diminution in prevalence, Epid. 111, 113
 — in London, prevalence stationary, Epid. 112, 118, 119
 — in Malta, greatest prevalence in rural districts, Epid. 104, 105, 111, 118, 119
 — in Nottingham, prevalent, Epid. 111, 115
 — in Wigan, prevalence stationary, Epid. 112, 114, 117, 118, 119
 — in Woolwich, reduction of death-rate, Epid. 126
 —, see also *Summer diarrhoea*
 DICKINSON, VINCENT.—Case of enlarged spleen, Child. 55; discussion (p. 56): Dr. Forsyth
 Diet in chronic nephritis, articles to be excluded, Therap. 143
 —, to be included, Therap. 143
 —, unnecessary restrictions, Therap. 143
 — in duodenal ulcer, Surg. 91, 100
 — in relation to thyroid gland, Baln. 10
 — in treatment of acute gastric ulcer, Therap. 126, 127, 134
 —, of chronic gastric ulcer, Therap. 128, 130
 —, quality, Therap. 130
 — (special), analysis of feces in diseases of pancreas under, Med. 204, 205, 206
 Dietetic rules for patients suffering from skin diseases, Baln. 77

- Dieulafoy, tuberculous infection of tonsils, quoted, Child. 30
 Digestion, effect on vaccine, R.S.M. 91
 Digestive ferments, action on bacteria, R.S.M. 93
 Dihydrogen sodium phosphate, anhydrous salts in, Med. 16, 17
 ————, preparation of, Med. 16, 17
 Dilatation, atonic, of stomach, fats in treatment of, Med. 33, 34
 Diphtheria, diagnosis, defects in, R.S.M. 9, 10
 ———, hæmorrhagic, diagnosis of purpura fulminans from, Child. 77
 ———, malignant, in infant, case of (J. D. Rolleston), Child. 74
 ———, epidemics recorded, Child. 75
 ——— and tracheotomy, complete stenosis of larynx left after, completely cured by repeated intubations spread over three years (StC. Thomson), Laryng. 35
 ———, treatment by antitoxin, defects in, R.S.M. 10
 ———, limitations of, R.S.M. 213
 Diphtheritic membrane, presence of, in trachea, after death, of boy who died apparently from uræmia (J. Walter Carr), Child. 90
 Diplegia (spastic) in case of mental defects, Child. 70
 Disease, cause of intense poverty, Epid. 7
 ———, classes of, responding most readily to poverty, Epid. 18
 ——— (chronic), condition of mouth in (R. Ackerley), Odont. 94-103
 ———, incidence of, heaviest among poorest classes, Epid. 11
 ———, poverty cause of, Epid. 7
 ———, prevention of, necessary part of cure, Child. 34
 ———, public assistance in prevention of, Epid. 42, 43
 ———, reduction of, through agency of reduction of poverty, Epid. 34
 ———, relation of poverty to, in Manchester, Epid. 15
 Diseases (communicable), characteristics of, Epid. 99
 ———, names of, substitution of names of micro-organisms for, R.S.M. 142
 Disinfection, methods of, in isolation hospitals, Epid. 84, 85
 Dislocation, recurring, of humerus, detachment of tendon of subscapularis muscle for (W. G. Spencer), Clin. 20
 ——— of shoulder, treatment by static wave current, Electr. 107
 Diuresis, sign of increased thyroid activity, Baln. 9
 Diuretic action of weaker saline waters, Baln. 80, 84
 Diuretics, action of some (H. H. Dale and P. P. Laidlaw), Therap. 38
 ——— in treatment of acute nephritis, difficult, Therap. 145
 ——— of chronic nephritis, how far desirable, Therap. 144, 145
 Diverticulum, see *Meckel's diverticulum*
 DIXON, Professor W. E.—Discussion on teaching of therapeutics in hospital wards, Therap. 12
 ———, discussion on treatment of gastric ulcer, Therap. 133
 ———, laboratory report on kangalugi root, Therap. 68
 ———, proprietary, patent, and secret remedies; an address introductory to a discussion on the subject, Therap. 82; discussion, renewed (pp. 91-100): Mr. Beddoes, Mr. Gamble, Dr. Duncanson, Dr. Martin, Mr. Woolcock, Mr. Harrison, Professor Cushny, Professor Dixon (reply)
 ———, researches on pineal gland alluded to, Neur. 76
 Döderlein's method of pubiotomy, Obst. 121
 Does (pregnant), grafting of foetal bones into, Path. 132
 Dogs, extirpation of pancreas in, Path. 88
 ———, production of acute and subacute pancreatitis in, by injection of turpentine, Path. 80, 81, 89
 ———, of chronic pancreatitis in, by introduction of intestinal bacteria from duodenum into pancreatic ducts, Path. 89, 90
 Domestic duties, ignorance of, among mothers in poorest classes in towns, Epid. 5, 6
 DONELAN, J.—Discussion on case of large chronic swelling in tonsillar region, Laryng. 133

- DONELAN, J.—Discussion on case of leukoplakia of soft palate, Laryng. 83
 —, discussion on case of malformation of soft palate and uvula, Laryng. 131
 — of secondary specific pharyngitis, Laryng. 28
 — on model demonstrating "back" and "belly" breathing, Laryng. 50
 — on probable late secondary specific pharyngitis, Laryng. 155
 — on sanatorium, silence and galvano-cautery treatment of tuberculosis of throat, Laryng. 10
 — on thyroid gland operation in extra-laryngeal inoperable carcinoma, Laryng. 16
 — on treatment of case of tuberculosis of the larynx, Laryng. 153
 — of tuberculous laryngitis by galvano-puncture, Laryng. 122
 — on tuberculosis of larynx in girl, Laryng. 128
 — on vulsellum-catch forceps, Laryng. 125
 —, extension of the cuticle inside the nostrils of a man aged 41, Laryng. 90; discussion (p. 90): Dr. Paterson, Dr. Pegler
 —, extensive fracture of nasal septum, complicating maxillary suppuration, in a woman aged 39; operations; recovery, Laryng. 89
 —, specimen of goitre and portions of larynx and trachea from a woman aged 37, Laryng. 38; discussion (p. 38): Dr. H. FitzGerald Powell
 — tuberculous ulceration of epiglottis apparently healed by frequent use of electro-cautery, Laryng. 156
 —, two cases shown, Laryng. 134
 DORAN, ALBAN.—Discussion on case of cystic tumour of right broad ligament, Obst. 70
 —, discussion on case of primary carcinoma of Fallopian tube, Obst. 77
 — on case of tubal mole, Obst. 238
 — on double uterus with dysmenorrhoea, Obst. 242
 DORE, S. E.—Hypertrophic lichen planus of two months' duration in a boy aged 10, Derm. 16
 —, linear naevus of unusual character in a boy aged 18, Derm. 86
 —, lupus vulgaris erythematodes, Derm. 97
 —, monilithrix in a boy aged 3 years and 9 months, Derm. 85
 —, ringworm affecting the upper lip as well as the beard region and hand in a man aged 28, Derm. 111
 —, and MORRIS, Sir MALCOLM.—An unusual condition of the lips, Derm. 18; discussion (p. 19): Dr. Fox, Dr. Pringle
 —, curious affection of the finger- and toe-nails, Derm. 6; discussion (p. 7): Dr. Dore, Dr. Colcott Fox, Dr. Whitfield, Dr. Pringle, Dr. G. Pernet
 —, pityriasis rubra in a man aged 21, Derm. 60; discussion (p. 61): Dr. Galloway
 —, report on case of extensive acne with scars, Derm. 7
 —, thickening of the skin of the palms and soles in a woman aged 31, Derm. 6
 Douches, general, stimulation of cutaneous surface by, Baln. 10
 Dowd, reappearance of malignant growth subsequent to gastro-enterostomy, Surg. 139
 DOWSETT, E. B.—Discussion on spindles of von Ebner, Odont. 150
 Doyme, R. W., first description of discoid familial, or "Coppock" cataract, Child. 68, 69, 70
 Drainage, peritoneal, and omentopexy, for chronic ascites connected with hepatic cirrhosis, patient shown a few months after (F. Parkes Weber), Clin. 80
 DREW, DOUGLAS.—Discussion on case of multiple cystic hygroma of neck, Child. 157
 — on diagnosis and treatment of intussusception in children, Child. 173
 —, separation and displacement forwards of the lower epiphysis of the femur treated by plates and screws, Child. 56; discussion (p. 57): Mr. Drew, Mr. A. R. Thompson, Mr. Lockhart Mummery, Mr. Drew (reply)
 Dropsy, apocynum administered for, Therap. 38, 39
 —, non-inflammatory serous pericardial effusion in, Med. 56
 Drugs, nomenclature, confusion in, Therap. 90
 DUCKWORTH, Sir DYCE, Bart.—Case of gout, with large tophaceous deposits, in a Mahomedan, Clin. 21

- DUCKWORTH, Sir DYCE, Bart.—Case of gout, with uratic tophi in a boy aged 14, Clin. 23;
discussion (p. 24): Dr. Philip G. Lee
- , discussion on case of pigmentation of circum-oral skin and of buccal mucosa, Clin. 9
- on case of rheumatoid arthritis with albuminuria, and with great enlargement of liver, Clin. 62
- on cases of adiposis dolorosa, Clin. 60
- on teaching of therapeutics in hospital wards, Therap. 14
- on rheumatoid arthritis with enlargement of lymphatic glands and spleen, Clin. 19
- DUDGEON, L. S.—On fatty degeneration of heart, quoted, Child. 82, 83
- , SHATTOCK, S. G., and SELIGMANN, C. G.—Attempts to produce chondromatous or osteomatous growths by the grafting of foetal bones, Path. 127-140
- Duffin, A. B., and Ferrier, D., F.R.S.—Glioma of pineal body, case quoted, Neur. 70, 71
- Dührssen, treatment of puerperal eclampsia, Obst. 200, 201
- DUNBAR, J. J. M.—Discussion on case of sarcoma of the ear and mastoid region, Clin. 164
- Duncan, J. Matthews, investigations as to puerperal death-rate, quoted, Obst. 232
- DUNCANSON, J. G.—Discussion on criticism of some principles in treatment of chronic nephritis, Therap. 146
- , discussion on proprietary, patent and secret remedies, Therap. 94
- on treatment of incipient pulmonary tuberculosis by antiseptic inhalations, Therap. 36
- Dunlop, association of dyspepsia with oxalate urinary deposit, quoted, Med. 3
- , solution of calcium oxalate in urine, alluded to, Med. 2, 3
- Duodenum, introduction of intestinal bacteria from, into pancreatic duct producing chronic pancreatitis in dogs, Path. 83, 90
- , stenosis of, caused by ulcer, Surg. 185
- , ulcer of, acid dyspepsia and, Surg. 74
- age incidence, Surg. 69, 79, 80, 92
- , and gastric hypersecretion, Surg. 181, 182, 184, 185, 186
- , cardiospasm in, Surg. 81
- , cases operated upon by Mr. Moynihan (1900-1908), analysis (H. Collinson), Surg. 79
- , cause of hæmorrhage in, Surg. 195, 197
- , chill as cause of, Surg. 87, 106
- , chronic, analysis of cases operated on, Surg. 80
- , —, —, methods of operation, Surg. 81
- , —, pancreatitis in, Med. 172
- , —, treatment, surgical, recommended, Surg. 109
- , diagnosis (B. G. A. Moynihan), Surg. 69
- , —, by administration of mixed carbonates, Surg. 88, 89
- , —, difficulty of, Surg. 92, 93, 94, 95, 111
- , —, from appendicitis, Surg. 115
- , —, from gall-stones, Surg. 88, 89, 90, 92
- , —, from gastric crises, Surg. 107
- , —, methods of examination, Surg. 96
- , —, value of anamnesis in, Surg. 72, 73, 115
- , disease of appendix diagnosed as, Surg. 202, 203
- , fats in treatment of, Med. 32, 34
- , gastric stasis in, Surg. 73
- , gastric ulcer co-existent with, Surg. 92
- , —, mistaken for, Surg. 93, 94
- , hæmorrhage in, Surg. 74, 76
- , —, occult, Surg. 76, 112
- , —, occurring as hæmatemesis or mælena, Surg. 75-77, 80, 93, 99, 100, 103
- , healing, Surg. 103

- Duodenum, ulcer of, hunger-pain in, Surg. 70, 94, 97, 98, 99, 100, 102, 112
 ———, in childhood, Surg. 92
 ———, local tenderness in, Surg. 90
 ———, pain in, Surg., 70, 71, 87, 88, 93
 ———, appetite during, Surg. 71
 ———, onset, Surg. 102
 ———, relief of, Surg. 87, 88
 ———, by taking food, Surg. 70
 ———, perforating, Surg. 113
 ———, analysis of cases operated on, Surg. 79
 ———, post-operative vomiting, Surg. 86
 ———, prophylaxis by diminution of secretion of hydrochloric acid, Surg. 105
 ———, pyloric spasm reflex and protective in, Surg. 73
 ———, relation of hyperchlorhydria to, Surg. 99, 101, 108
 ———, sex-incidence, Surg. 69, 79, 80, 111, 204
 ———, statistics, Surg. 89
 ———, symptoms, Surg. 69-77, 112
 ———, absence of physical signs, Surg. 73
 ———, test meals in, Surg. 74, 92, 112
 ———, tetany in, Surg. 81
 ———, treatment (B. G. A. Moynihan), Surg. 69
 ———, care of teeth important in, Surg. 105
 ———, dietetic, Surg. 91, 100
 ———, medical, Surg. 91, 104, 105, 106
 ———, surgical, Surg. 77-79
 ———, by excision, Surg. 92
 ———, by gastro-enterostomy, Surg. 78, 79, 81, 92, 93, 95, 116
 ———, by gastro-jejunostomy, Surg. 109, 110
 ———, infolding ulcer in, Surg. 79, 96
 ———, methods, Surg. 78, 79, 81, 92, 93, 95
 — and stomach, division of, sixth step in operation for removal of lymphatic area connected with stomach, Surg. 153
 ———, primary hyperplastic tuberculosis of (W. G. Nash), Clin. 40
 Dupuytren's contraction, treatment by fibrolysin, Clin. 89
 Dust, agency of, in spreading summer diarrhoea, Epid. 139, 140
 ———, relation of sporadic scarlet fever to, Epid. 78, 90
 Dysentery, general paralytics easily succumb to, Neur. 49
 ———, in relation to infective arthritis, Baln. 93
 Dysmenorrhœa in case of double uterus, Obst. 242 (A. H. N. Lewers), Obst. 241
 ———, treatment by electricity, Electr. 7, 8-15, 18, 19, 26; Obst. 21, 27
 ———, by static wave current, Electr. 100
 Dysotocia, explanation of, Obst. 5
 Dyspepsia (acid), Surg. 112
 ——— and duodenal ulcer, Surg. 72, 74, 107
 ——— and hypersecretion, Surg. 206
 ——— (atonic), cause of oxaluria, Med. 3, 7, 8, 21
 ——— in early phthisis, R.S.M. 17
 ———, importance of examination of teeth, Odont. 95
 ———, in relation to condition of teeth, Odont. 102
 ———, nervous, Surg. 184
 ———, fats in treatment of, Med. 33, 34
 ———, result of gastric hypersecretion, Surg. 180
 ———, rôle of fats in treatment of, Med. 25
 ———, with gastric dilatation, relieved by static wave current, Electr. 104
 ———, see also *Appendix dyspepsia*

- Dysphagia, considerable, occurring in tuberculosis of larynx, treated and relieved by congestion hyperæmia (W. Howarth), *Laryng.* 164
 — in tuberculous laryngitis, treatment with injections of alcohol, *Clin.* 201
 Dyspnoea accompanying loss of voice (W. Stuart-Low), *Laryng.* 162
 — swelling on left side of neck (N. Patterson), *Laryng.* 22
- Ear, auditory discharges from organic disease, *Neur.* 5
 — of deaf-mutes, pathological changes found in, lantern-slide demonstration (A. A. Gray), *Otol.* 11
 —, drum of, normal, adventitious membrane, formed after radical mastoid operation, resembling (H. J. Davis), *Otol.* 58
 —, internal, normal anatomy, specimen illustrating (S. R. Scott), *Otol.* 23
 —, left, objective clicking sound in (H. Tilley), *Otol.* 36
 — — and rhythmical "clicking" noise in, associated with synchronous muscular contraction of certain muscles forming floor of mouth (H. Tilley), *Clin.* 93
 — (middle), disease of, diffuse basal lepto-meningitis complicating, preceded by labyrinthitis, *Otol.* 26
 — —, treatment by vaccine, *R.S.M.* 160
 — —, epithelioma of, case operated on six times, *Otol.* 30
 — —, mild catarrh of, previous to left facial paralysis (H. J. Davis), *Otol.* 60
 — —, muco-pus in, of infants dying from various disorders, *Otol.* 26, 27
 — —, relation of cerebellar abscess to, *Otol.* 23, 26
 — —, streptococcal infection, treatment by streptococcus vaccine, *R.S.M.* 67, 68
 — —, suppuration, chronic, twenty specimens of, and sequelæ, eighteen of the bones being of the infantile types and two cellular (A. H. Cheatile), *Otol.* 41
 —, right, deafness and discomfort in, as early symptoms in case of epithelioma originating near right Eustachian tube (E. Law), *Otol.* 28
 —, sarcoma of, radium treatment (N. S. Finzi), *Clin.* 157
 East London Hospital, Shadwell, statistics of pericardial effusion from (J. S. Pearson), *Med.* 124
 Eaux Bonnes, cure of female sterility at explained, *Baln.* 10
 "Ebers Papyri," quoted, *Odont.* 155
 von Ebner, spindles of (D. E. Caush), *Odont.* 144
 ECCLES, W. McADAM.—Discussion on X-ray treatment of lymphadenoma, *Electr.* 135
 Ectasia, three forms of, *Electr.* 113
 Ectopia vesicæ, prolapse of intestine through (A. C. D. Firth), *Child.* 55
 Eczema, due to excessive ingestion of salt in food, *Baln.* 76, 77
 —, treatment by application of violet-flame discharge, *Electr.* 77
 — — by vaccine, *R.S.M.* 103, 183
 EDEN, T. W.—Discussion on leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, *Obst.* 49
 —, discussion on modern methods of delivery in contracted pelvis, *Obst.* 125
 — — on slighter forms of puerperal sepsis, *Obst.* 233
 EDMUNDS, WALTER.—Action of thyroid and antithyroid preparations, *Path.* 100
 Edmundsthal Heilstätte, Hamburg, results of tuberculin treatment in minute doses satisfactory at, *R.S.M.* 116
 Education (compulsory), extension of age-limit, *Epid.* 41
 —, disappointing results of, *Epid.* 38, 39
 Egg-albumin in diet, albuminuria of nephritis not increased by, *Therap.* 139
 Egypt and North Africa, diseases influenced beneficially by climate of, *Baln.* 126
 — —, influence of climate of, on disease (H. E. L. Canney), *Baln.* 115
 — —, risk of "chill" in, *Baln.* 125
 Egyptians (Ancient), dentistry among, *Odont.* 154, 155
 — —, skulls of, exhibited (A. S. Underwood), *Odont.* 153

- Ehrlich, side-chain of, R.S.M. 196
 ————, detachment, R.S.M. 156
 Ehrlich's diazo-reaction, R.S.M. 202
 Electric currents, continuous application of, in prevention of talipes, Electr. 105
 ———— (direct), in gynaecological affections (J. C. Webb), Electr. 16
 ———— ionization with, Electr. 105
 ————, valuable, in treatment of sciatica, Electr. 105
 ————, kinds used in gynaecological disorders, Obst. 19
 ———— shock (20,000 volts), histological changes in brain from case of (F. W. Mott, F.R.S., and E. Schuster), Path. 140
 ———— stimulation of the heart, Anæsth. 5
 ———— wind (silent discharge) in static electricity, Electr. 75, 76
 ————, formation of ozone under, Electr. 76, 83, 84
 ————, therapeutic applications, Electr. 76
 Electricity, atmospheric (G. Mahomed), Baln. 49-58
 ————, current of, conveyance of micro-organisms along, R.S.M. 156
 ————, opposite charges of, carried by toxins and anti-toxins, R.S.M. 156
 ————, in production or control of auto-vaccination, R.S.M. 157
 ————, treatment by, in relief of symptoms of prostatic enlargement (J. S. Bolton), Electr. 141-146
 ———— (static), advantages of, Electr. 97
 ————, discharges, Electr. 75
 ————, disuse of, Electr. 97
 ————, nature of current, Electr. 80
 ————, passage of ions in, Electr. 75
 ————, production of, method, Electr. 74, 75
 ————, source of Röntgen rays, Electr. 81
 ————, stimulation of nervous system and muscles by, Electr. 80
 ————, therapeutic applications in treatment of psoriasis, Electr. 82, 83
 ————, of writer's cramp, Electr. 81
 ————, producing contractions of muscles, Electr. 82
 ————, recto-abdominal in treatment of obstinate constipation, Electr. 82
 ————, relief of locomotor ataxy, Electr. 84
 ————, with vacuum electrode, Electr. 82, 85
 ————, use of (J. A. Voorthuis), Electr. 73-81
 ————, in treatment of lupus, Electr. 73, 74, 78-80
 ————, see also *Spark application*
 ————, see also *Influence machine*
 ————, see also *Morton wave current*
 Electro-cautery in gynaecology, risks of, Obst. 27
 ————, tuberculous ulceration of epiglottis apparently healed by frequent use of (J. Donelan), Laryng. 156
 ———— and refrigeration, in treatment of naevi (E. R. Morton), Electr. 37
 Electro-static machines in use in United States, Electr. 83, 84, 85
 Electro-therapeutics, in gynaecology (S. Sloan), Electr. 1
 ————, in gynaecology, origin of, Electr. 4
 Electro-theraputists, clinical knowledge important to, Electr. 146
 Electro-vaccination in treatment of rodent ulcer, R.S.M. 155
 ———— of skin diseases, R.S.M. 153-155, 158
 Electrode (rectal), application to prostate for administration of high-frequency currents, Electr. 142, 147
 ————, pattern required, Electr. 142
 ————, relief of atony of bladder by application of, Electr. 147
 ———— with static wave current in treatment of chronic constipation, Electr. 102
 ———— (urethral), passage in diseases of prostate not recommended, Electr. 147

- Electrode (uterine), Obst. 16, 17, 18
 — (vacuum), in treatment of gonorrhœa, Electr. 148
 Electrodes of electro-static influence machines, Electr. 75
 — — — — —, effects of separation, Electr. 75
 Electrolysis in treatment of nævi, Electr. 38, 46
 — — — — — of uterine tumours, method of Apostoli scientific, Electr. 4, 5
 Elephantiasis associated with tertiary syphilis (H. G. Adamson), Derm. 81
 — — — — — scroti, Surg. 171
 ELMSLIE, R. C.—Fracture of the humerus at the site of an innocent cyst, Clin. 192; discussion (p. 194): Mr. A. Pearce Gould, Dr. A. C. Jordan, Mr. Lockhart Mummery
 — — — — —, three cases of enlargement of tubercle of the tibia (Schlatter's disease), Clin. 189; discussion (p. 191): Mr. Pearce Gould, Mr. Cyril Nitch, Mr. Elmslie (reply)
 Emaciation, acute, following the formation of biliary fistula, treated by cholecyst-duodenostomy (J. D. Malcolm), Clin. 83
 — — — — —, constancy of, in cancer of stomach, Surg. 133
 Embolism, pulmonary, risk of following calomel injection in syphilis, Surg. 213
 Embolus, presence of, in diagnosis of endocarditis, Therap. 101, 102
 Embryo, human, 16 mm., auditory apparatus in, shown in reconstruction model (by wax-plate method of Born) (G. J. Jenkins), Otol. 34
 EMERY, W. D'ESTE.—Discussion on vaccine therapy, R.S.M. 187
 — — — — —, pathology of acne, quoted, Path. 174
 Empirical remedies, over-use of, Therap. 7
Empusa musca, destruction of house-flies by, Epid. 154, 156, 166
 Empyema, acute, in adults, following acute pneumonia or pleurisy, prognosis, Surg. 55-61
 — — — — —, of infancy, prognosis in, Surg. 53
 — — — — — (left-sided), diagnosis of purulent pericarditis from, Med. 66
 — — — — — (pneumococcal), prognosis, Med. 66
 — — — — — (pulsating), Med. 61, 75, 76
 — — — — —, purulent pericardial effusion associated with, Med. 126-128
 — — — — —, subjects of, in childhood, insurability of lives, Surg. 55
 — — — — —, treatment by tapping and incision, Clin. 121
 — — — — — by thoracoplasty in two operations (J. E. Adams), Clin. 90
 — — — — — by vaccine inoculation, R.S.M. 58
 — — — — — (tuberculous), prognosis, Surg. 61
 — — — — —, with purulent pericarditis, Med. 79
 Enamel, chalkiness of, early stage of decay, Odont. 72, 74, 75, 83, 84
 — — — — —, chemico-parasitic processes acting upon, Odont. 81
 — — — — —, dental, power of resistance to acids of decay, Odont. 78, 81, 83
 — — — — —, sole barrier against acids of decay, Odont. 73
 — — — — —, hypoplasia of, Odont. 79, 82, 84
 — — — — —, connexion with malnutrition in infancy, Odont. 79, 82, 84
 — — — — —, resistivity to caries, Odont. 81, 83
 — — — — —, susceptibility of, to acids producing decay, Odont. 79
 Enamel organ, histology of, Odont. 127
 Encephalitis, acute, Neur. 132
 — — — — —, complicating influenza, Neur. 132
 Enchondroma, subglottic, laryngo-fissure for (StC. Thomson), Laryng. 13
 Endaneurysmorrhaphy, obliterative (Matas's operation), for popliteal aneurysm (Maynard Smith), Clin. 130
 Endocarditis, acute, diagnosis, Therap. 101
 — — — — — (infective), definition, Therap. 114, 116
 — — — — —, in relation to chorea, Therap. 101
 — — — — — to rheumatism, Therap. 101, 102
 — — — — —, treatment by collargol, Therap. 105, 108, 112, 115, 116
 — — — — — by quinine, Therap. 114

- Endocarditis (infective), treatment by serum, Therap. 104, 105, 114, 115
 ——— by vaccine, Therap. 104, 105, 114, 115
 ———, reason for failure, R.S.M. 119
 ——— (malignant), case of agglutination of streptococcus, R.S.M. 118
 ———, treatment by oral administration of streptococcus vaccine, R.S.M. 117, 118
 ——— of tricuspid valve, in child aged 6 (F. H. Hawkins), Child. 139
 ———, treatment of (J. Cowan), Therap. 101; discussion (pp. 113-116): Dr. J. Mackenzie, Dr. Sainsbury, Dr. Morison, Dr. Pitt, Dr. Cowan (reply)
 ———, with rheumatism, treatment by salicylate, Therap. 104
 Endocardium, nodules on, shown in heart of infant (R. C. Jewesbury), Child. 45
 Endometritis, treatment by electricity, Obst. 26
 ———, with metritis, in slight puerperal sepsis, treatment, Obst. 228, 233
 ——— (septic), treatment by electricity, Electr. 7, 8, 10-20, 23
 ——— with curette, reasons against, Obst. 233
 ———, with metritis, in slight puerperal sepsis, Obst. 224, 225
 Endometrium, decidual reaction extending from, to tubal mucosa, cause of tubal pregnancy, Obst. 238
 Endo-skeleton in relation to development of neoplasms, Laryng. 159
 Endothelioma of ethmoid, case (C. Nourse), Laryng. 62
 ———, microscopic report on specimen (V. Wyatt Wingrave), Laryng. 63
 ———, operation for removal, Laryng. 63
 ———, removal, methods discussed, Laryng. 65-68
 ——— of temporal bone, case of (J. Dundas Grant), Otol. 32
 ——— ——— (D. R. Paterson), Otol. 61-62
 ——— of tongue, in cases of rare tumours of base of tongue (F. Eve), Clin. 173
 Endotoxin (tubercle), injection of, effect on opsonizing action of serum of healthy rabbits (R. Tanner Hewlett), Path. 165
 England, urban districts, diarrhoea most prevalent in, Epid. 104
 ——— and Wales, phthisis mortality, maximum incidence of rate in males and females in urban and rural counties compared, Epid. 28
Enhydrina valakadien (common seasnake), venom of, action on central nervous system, Neur. 106
 Eno's fruit salt, analysis, Therap. 86
 Enterectomy in case of intussusception of small intestine containing sarcoma of intestinal wall (C. A. S. Ridout), Child. 161
 Enteritis, zymotic, Epid. 105, 107
 ———, see also *Diarrhoea*
 ———, aetiology, Epid. 123
 ———, notification of, Epid. 125
 Enteroptosis, due to belly-breathing, Laryng. 49
 Enterostomy in intussusception, Child. 169
 Enuria, bacterial infection in, R.S.M. 18
 Eosinophile cells in exudate from tick-bites on horse (J. B. Cleland), Path. 42
 Ependymitis, or posterior basic meningitis, followed by internal hydrocephalus and amaurosis, without definite ophthalmoscopic changes (F. Parkes Weber), Child. 62
 Epidemics, case incidence of measles at public school with notes on pre-eruptive symptoms (H. G. Armstrong), Epid. 45
 ———, scarlet fever; its home treatment and prevention (R. Milne), Epid. 57
 Epidemiology, experimental or comparative, Epid. 219
 Epidermolysis bullosa, case of (G. Norman Meachen), Derm. 74
 Epididymitis (tuberculous), case, treatment by vaccine inoculation, apparent failure, R.S.M. 35
 Epigastric region, pain in, Obst. 8
 Epigastrium, bulging or prominence in which pulsation may be felt (Auenbrugger's sign) in pericardial effusion, Med. 58, 62
 ———, incision in, operations on pericardium by way of, Med. 91, 92

- Epigastrium, systolic recession in, Med. 58
- Epiglottis, fauces, hard palate and nose, lupus of (W. Stuart-Low), Laryng. 86
- , fungating mass on, with implication of larynx (H. J. Davis), Laryng. 140
- , left ary-epiglottic fold, and left inter-arytenoid space, tuberculosis of, healed by galvano-cautery and sanatorium treatment (StC. Thomson), Laryng. 7
- , lupus of, treatment by galvano-cautery, Laryng. 87
- , tuberculosis of, treated by amputation of diseased portion (H. Tilley), Laryng. 32
- , tuberculous disease of (W. H. Kelson), Laryng. 112
- , —, ulceration of, apparently healed by frequent use of electro-cautery, Laryng. 156
- , ulceration of, probably epitheliomatous (H. Barwell), Laryng. 145
- Epilepsy and special sense discharges from organic disease, Neur. 1
- , association with lymphatism, Anæsth. 53, 56
- , general, case of trephining for, shown after interval of three years (W. G. Spencer), Clin. 65
- (Jacksonian), following syphilis, Neur. 64
- , possibility of bacterial infection in, R.S.M. 17
- (syphilitic), Neur. 52, 62, 63, 64
- , uncinæ, Neur. 4
- , see also *Convulsions, epileptiform*
- Epileptiform attacks accompanying laryngeal vertigo, Laryng. 81
- Epiphyseal line, direction of, Child. 57
- Epiphysis (lower) of femur, separation and displacement forwards, treatment by plate and screws (D. Drew), Child. 56
- Epistaxis, with hereditary multiple telangiectases, Clin. 109
- Epithelial pearls (cell-nests), Path. 152
- Epithelioma accompanying lupus erythematosus (J. H. Sequeira), Derm. 100
- , originating near right Eustachian tube, case of, with deafness and discomfort in right ear as early symptoms (E. Law), Otol. 28
- , protective value of inoculation of spleen tissue against, Therap. 76
- of larynx (E. A. Peters), Laryng. 140
- , —, four cases shown after operation (StClair Thomson), Clin. 115
- , —, in man aged 69, fourteen months after operation (C. Nourse), Laryng. 143
- of left vocal cord, removal by thyrotomy (J. Dundas Grant and D. McKenzie), Laryng. 19
- , —, specimen of larynx showing fresh outbreak on, after removal of right cord for epithelioma, September, 1896 (H. Tilley), Laryng. 33
- of middle ear, case operated on six times, Otol. 30
- of nasal vestibules and adjacent areas, recurrence after surgical removal, radium treatment commenced (W. Hill), Laryng. 26
- of right vocal cord, case shown seven months after operation, no recurrence, development of cicatricial substitute for vocal cord (J. Dundas Grant), Laryng. 156
- , —, removal by thyrotomy (J. Dundas Grant), Laryng. 17
- of tongue without keratinization (F. S. Eve), Clin. 180
- , treatment by radium, Electr. 63
- Epithelium, metaplasia of, how produced, Otol. 66, 68
- Eppinger, catarrhal jaundice, quoted, Med. 171
- Erb, cases illustrating effects of syphilitic virus on nervous system, quoted, Neur. 43
- Error (functional), in bacteriological reports, R.S.M. 7
- , —, definition, R.S.M. 28
- , —, in diagnosis of cases, R.S.M. 6
- , —, mathematical limit of, R.S.M. 28
- , —, (working), R.S.M. 28, 29
- Eruption, acute and very extensive, of type of erythema iris (E. G. Graham Little), Derm. 2
- (cutaneous, syphilitic), children born with, probably immune to re-infection, Neur. 46
- , —, how produced, Neur. 36
- , —, relation to syphilitic meningitis, Neur. 36

- Erysipelas, accidental, curative effect on inoperable malignant tumours, Surg. 2, 9
 —, intercurrent (J. Galloway), Clin. 135
 —, seasonal prevalence, Epid. 92
 — of scrotum, as complication of rupture, Surg. 172, 177
 Erythema, peculiar, of left palate in man who had been considerable smoker (J. Dundas Grant), Laryng. 27
 — iris, acute and very extensive eruption of type of (E. G. Graham Little), Derm. 2
 — associated with lupus erythematosus (J. H. Sequeira), Derm. 94
 — nodosum, syphilitic (J. E. R. McDonagh), Derm. 50
 Ether, administration, age-limit for non-existent, Anæsth. 64, 65
 — by Clover's inhaler with bag, dangerous, Clin. 215, 216
 — in experiments on anaphylaxis, Obst. 9
 — of, by purely open method, apparatus for (R. H. Hodgson), Anæsth. 16
 —, dangerous to subjects of renal inadequacy, Anæsth. 69
 — under what circumstances dangerous, Anæsth. 71
 —, when suitable for children, Anæsth. 65
 —, dangers of, to alcoholics, Anæsth. 69
 —, death under, in cases of lymphatism not recorded, Anæsth. 49
 — followed by chloroform, routine anæsthetic for young children, Anæsth. 65, 66
 — inhalation, combined with drugs for pulmonary tuberculosis, Anæsth. 17
 —, without admixture of exhalations from lungs in treatment of pulmonary tuberculosis and other diseases (R. H. Hodgson), Clin. 213
 —, operation under, for case of lymphatism and exophthalmic goitre, Anæsth. 49
 — preceded by nitrous oxide, routine anæsthetic for youth and middle life, Anæsth. 66
 — preference over chloroform in secondary operations, Obst. 9
 — (pure) and hexamethylene-tetramin, inhalations of, in treatment of laryngeal tuberculosis, Laryng. 39
 Etherization, rectal, when to be employed, Anæsth. 71
 Ethmoid region, endothelioma of (C. Nourse), Laryng. 62
 —, case, microscopical report on specimen, Laryng. 63
 —, operation for removal, Laryng. 63
 —, removal, methods discussed (Jensen's method, modified, Denker's operation, Rouge's operation), Laryng. 65-68
 —, preliminary laryngotomy, Laryng. 67
 Ethyl, chloride of, administration of, to children, Anæsth. 65
 —, followed by ether associated with oxygen, anæsthetic recommended for asthenics, Anæsth. 66
 —, uncertainty of effects on alcoholics, Anæsth. 68
 — spray in treatment of lupus vulgaris, Electr. 42
 Eucalyptus oil rubbing in scarlet fever, Epid. 57
 Eunuchs (Chinese), generative organs and pelvis of, Path. 103, 124-126
 — local obesity of, Path. 122
 —, measurements of, Path. 106
 —, greater in lower limbs, Path. 106
 —, pelvis in, measurements, Path. 120-124
 —, width, excessive, Path. 121
 —, rapid growth at time of puberty, Path. 106
 Eustachian tube, mucous polypus presenting at pharyngeal orifice of, in case of bilateral chronic adhesive otitis media (J. Arnold Jones), Otol. 55
 — and pharynx, bands between (E. A. Peters), Laryng. 69
 —, right, epithelioma originating near, case of, having deafness and discomfort in right ear as early symptoms (E. Law), Otol. 28
 EVANS, A.—Discussion on epithelioma of larynx, Laryng. 141
 —, discussion on excision of entire tongue, Laryng. 139
 — party-wall pharyngeal cancer, Laryng. 142

- EVANS, A.—New growth of right tonsil invading the surrounding tissues, *Laryng.* 147;
discussion (p. 148): Dr. H. J. Davis, Mr. Evans (reply)
- EVANS, Miss B. W., observations on boarding out of pauper children referred to, *Epid.* 40
- EVANS, E.—Excision of the larynx, lower part of the pharynx, and upper end of the oesophagus
for malignant disease (squamous-celled carcinoma) of those structures, *Clin.* 44;
discussion (p. 47): Mr. C. Symonds
- EVANS, J. HOWELL.—Case of X-ray carcinoma, *Clin.* 145
- congenital hemidystrophy, *Child.* 23; discussion (p. 23): Dr. F. Parkes Weber, Mr.
Lockhart Mummery, Dr. Higgs
- , discussion on case for diagnosis, *Clin.* 148
- EVANS, WILLMOTT H.—Case of xeroderma pigmentosa with unusual features, *Derm.* 57;
discussion (p. 58): Dr. Colcott Fox, Dr. Pringle, Dr. F. Parkes Weber
- EVE, F. S.—Arterio-venous aneurysm with exophthalmos following an injury to the head,
Clin. 77
- , discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 93
- on treatment by radium of malignant growths, *Clin.* 166
- , partial luxation of the odontoid process, *Clin.* 78
- , three rare tumours of the base of the tongue (two of them endotheliomata) with remarks
on the surgery of malignant disease of the tongue, *Clin.* 173; discussion (p. 186): Dr.
Lazarus-Barlow, Mr. W. G. Spencer, Mr. Carless, Mr. Eve (reply)
- , transverse fracture of both patellæ, with much separation of fragments, treated by
operation, *Clin.* 78
- Evian, waters of, diuretic action explained, *Baln.* 9
- Ewald, appendicitis larvata, quoted, *Surg.* 187
- , fats in treatment of gastric disorders, *Med.* 25, 26, 27
- EWART, W.—Discussion on cases of lobar pneumonia with displacement of heart, *Clin.* 27
- , discussion on examination of intestines by X-rays, *Electr.* 122
- on pericardial effusion, *Med.* 73
- on treatment of incipient pulmonary tuberculosis by antiseptic inhalations,
Therap. 35
- on dorsal percussion of the thorax and of the stomach, and a new stomach sign, *Med.*
211-239; discussion (p. 239): Dr. Mitchell Bruce, Dr. Gossage, Dr. Ewart (reply)
- Ewing, Jones, nutritional requirements of cancer-cell, quoted, *Surg.* 11
- Exaltation in cerebral syphilis, *Neur.* 51
- in general paralysis, possible explanation, *Neur.* 48
- Excoriation, neurotic, *Derm.* 28
- , of "dug-out" type of Colcott Fox, multiple keloid associated with (H. G. Adamson),
Derm. 42
- Excretory organs, failure to deal with excess of chlorides, *Baln.* 78
- Exercise (severe), test of blood after, to determine extinction of bacterial infection, *R.S.M.*
24, 25
- , want of, in towns, ill-effects on children of poorest classes, *Epid.* 5
- Exhaustion, cause of death in tetanus, *Med.* 40
- Exophthalmos, associated with arterio-venous aneurysm following injury to head (F. S. Eve),
Clin. 77
- pulsating (G. Williams), *Clin.* 171
- Exostoses, multiple, case of (H. Burrows), *Child.* 164
- of mandible (F. Coleman), *Odont.* 21
- of right external meatus (H. Tod), *Otol.* 76
- Expression (facial), result of extraction of six-year-old molars on, *Odont.* 157
- Extremities, symmetrical trophic lesions in child; syringomyelia of Morvan's type (H. Morley
Fletcher), *Clin.* 98
- (lower) effect on, of ovarian tumour pressing on sacral nerves, *Obst.* 7
- chronic arteritis obliterans of, associated with phlebitis (F. Parkes Weber),
Clin. 97

- Eye, abnormal congenital pigmentation of one (N. B. Harman), Child. 67
 —, visual discharges from organic disease, Neur. 11
 EYRE, J. J.—The hygiology of Naples, Baln. 14
 EYRE, J. W. H.—Discussion on the bacteriology of pyorrhœa alveolaris, Odont. 68
 —, discussion on therapeutic value of lactic-acid bacillus, Therap. 59
 — on vaccine therapy, R.S.M. 60
 — and PAYNE, J. L.—Observations on the bacteriology of pyorrhœa alveolaris, and treatment of disease by bacterial vaccines, Odont. 29
- Face, excavating rodent ulcer of, under treatment by radium (C. J. Symonds), Clin. 74
 —, sensation in, altered in cases of central nervous disease, Neur. 87
 — and mouth (specimen) of female infant, showing arrest of development of right half of tongue, combined with cleft of soft palate and palato-lingual fold (A. R. Tweedie; pathological report by A. Keith), Laryng. 99
 Faeces, analyses of, diagnostic value in diseases of pancreas (P. J. Cammidge), Med. 185-202
 —, diarrhœal, contagiousness of, intense, Epid. 96
 — — —, proved by infection during laboratory examination, Epid. 97
 —, examination of Gram-stained cover-glass preparations to determine nature, Therap. 56
 — by Strasburger's fermentation apparatus, Therap. 55, 56, 57
 —, reaction of, test for distinguishing abnormal putrefaction of proteids from fermentation of carbohydrates in intestine, Therap. 55, 56, 57
 — in diseases of pancreas, analysis of, diagnostic value (P. J. Cammidge), Med. 185-202
 — — —, under special diet, Med. 204, 205, 206
 — — —, appearance, Med. 185
 — — —, bacteriological examination, Med. 202
 — — —, bile-pigment in, Med. 197
 — — —, blood in, Med. 199, 200
 — — —, chemical analysis, Med. 187
 — — —, fats, Med. 187-196
 — — —, total, Med. 187-191, 196
 — — —, inorganic ash in, Med. 196
 — — —, microscopical examination, Med. 186
 — — —, reaction, Med. 186
 FAGGE, C. H.—Discussion on anatomical and pathological preparations, Otol. 26
 —, discussion on case of endothelioma of temporal bone, Otol. 33
 — on early symptoms in case of epithelioma, originating near right Eustachian tube, Otol. 30
 — on Kuhn's instruments for per-oral intubation, Otol. 50
 — on large nasopharyngeal growth in a boy aged 12, Otol. 48
 — on pedunculated papilliform growth, Otol. 46
 — on thoracoplasty in treatment of empyema, Clin. 92
 Fallopian tube, carcinoma (primary) of, associated with acute inflammatory mischief (W. W. H. Tate), Obst. 75
 —, chorio-epithelioma primary in, Obst. 236, 237
 —, fibroma of, Obst. 239
 —, fibromyoma of, Obst. 239
 —, tuberculous adenoma of, with tuberculous salpingitis (J. Inglis Parsons and B. Glendining), Obst. 238
 —, see also *Mole (tubal); Pregnancy (tubal)*
 Faradaic current, harmful in acute sciatica, Electr. 105
 — in treatment of sclerodactyly, Clin. 32
 Farms, tuberculous, in Manchester milk-supply district, Epid. 225
 Fat intoxication, Anæsth. 50, 51
 Fatigue, estimation by opsonic index, R.S.M. 74

- Fats, digestion and absorption, interference of chronic pancreatitis with, Med. 190
 —, rôle of, in treatment of disorders of stomach (F. C. Moore and F. L. Ferguson), Med. 25
 — in faeces in diseases of pancreas, Med. 187-196
 — — — — —, relation of saponified to unsaponified, Med. 191
 — — — — — determined by distribution and activity of intestinal flora, Med. 193
- Fauces, epiglottitis, hard palate, nose, lupus of (W. Stuart-Low), Laryng. 86
 — and larynx, nasal cavities, tongue, cheek and lips affected by pemphigus vegetans (H. J. Davis), Laryng. 78
- FEDDEN, W. F.—Discussion on case of sarcoma of ear and mastoid region, treated by radium, Clin. 163
- Feeble-minded children, detention homes for, Epid. 41
- Feeding, method of, in acute gastric ulcer, Therap. 122
- Fehling, differentiation between male and female pelvis, alluded to, Path. 110
- Fehling's solution, reduction by urine in pityriasis rubra, Derm. 61
- Fellner, B., jun., combination of auscultatory and tactile methods of reading arterial pressure, quoted, Med. 209
- Females, among Skoptzi (Russian castrating sect), mutilation limited to ablation of mamme, Path. 118
- Femur, fracture, extracapsular, of neck in a boy (A. R. Thompson), Child. 22
 —, lower epiphysis of, separation and displacement forwards, treatment by plate and screws (D. Drew), Child. 56
 — (left), congenital absence of (J. P. Lockhart Mummery), Child. 101
 — and right humerus, shortening of, due to unobserved fractures in early life (H. Burrows), Child. 165
- FENWICK, W. SOLTAU.—Clinical significance of gastric hypersecretion and its connexion with latent disease of the appendix, Surg. 177-186
 —, symptoms of hypersecretion, Surg. 205
- FERGUSON, R. L., and MOORE, F. CRAVEN.—Rôle of fats in the treatment of disorders of the stomach, Med. 25; discussion (p. 35): Dr. Mitchell Bruce, Dr. Hertz, Dr. Saundby, Dr. Hutchison, Dr. Moore (reply)
- Ferrier, D., F.R.S., visual discharges from organic disease, quoted, Neur. 11
 —, and Duffin, A. B., glioma of pineal body, case quoted, Neur. 70, 71
- Fibrillary tremor ("delirium cordis" of McWilliam), Anaesth. 1, 5
- Fibrils of Betz cells, connexions discussed, Path. 29
- Fibro-caseous disease of both suprarenal capsules, extensive but incomplete, with absence of symptoms of Addison's disease (H. French), Path. 93
- Fibroid lung and displacement of lung (Sir J. F. H. Broadbent, Bart.), Clin. 133
 — tumour of cervix in vagina obstructing labour (W. S. A. Griffith), Obst. 188
 — — — — — and body of uterus (J. P. Hedley), Obst. 189
 — — — — —, very large, successful operation (A. E. Giles), Obst. 184
 — — — — —, large uterine, complicated by extensive retroperitoneal hæmatocele, operation, cure (W. Alexander), Obst. 139
 — — — — — (subperitoneal), necrosed and suppurating, treated by abdominal hysterectomy sixty-three hours after labour (P. D. Turner), Obst. 61
 — — in vaginal wall (H. Russell Andrews), Obst. 129
- Fibroid tumours treated by posterior Cæsarean section followed by total hysterectomy (H. R. Spencer), Obst. 82
 — — — — — by electricity, Electr. 7-20, 25
- Fibrolysin, mode of action unknown, R.S.M. 146
 —, therapeutic effects of, Clin. 89, 90
 —, therapeutical value proved, R.S.M. 146
 — in treatment of contracted joints and Dupuytren's contraction, Clin. 89, 90
 — — — — — of sciatica, Clin. 89
 — — — — — of stricture of rectum (H. C. Howard), Clin. 87

- Fibroma (soft, rapidly-growing), removal of, from posterior wall of left maxillary antrum by modified Rouge's operation (H. Tilley), Laryng. 96
- of Fallopian tube, Obst. 239
- Fibromyoma and pregnancy, thrombosis of veins and necrobiosis of tumour (T. G. Stevens), Obst. 54
- , degenerating, of cervix (F. McCann), Obst. 147
- of Fallopian tube, Obst. 239
- of uterus, alterations in menstruation, Obst. 213
- , complete disappearance of tumour, cases showing, Obst. 208
- , complicated by dilatation and hypertrophy of left heart, Obst. 215
- by valvular disease, Obst. 215
- , condition of patients prior to and after menopause, Obst. 218
- , growth of tumour to maximum, Obst. 213
- , life-history of (A. J. Wallace), Obst. 208-220
- , table of cases illustrating, Obst. 216, 217
- , parity of patients, Obst. 213
- , shrinking of tumour, cases showing, Obst. 211, 214
- , relation to menopause, Obst. 214, 219
- , palliative treatment not recommended, Obst. 219
- Fibrosarcoma of nose removed after temporary ligation of both external carotid arteries and laryngotomy (W. D. Harmer), Clin. 122
- Fibrosis of uterus, causing persistent menorrhagia (J. Inglis Parsons), Obst. 240
- Fibrous tissue surrounding dentigerous cysts, Odont. 159, 160
- Film-holder, sterilizable (Charles A. Clark), Odont. 85
- Film radiographs, method of ascertaining relative position of unerupted teeth by (C. A. Clark), Odont. 87
- Finger- and toe-nails, curious affection of (Sir M. Morris and S. E. Dore), Derm. 6
- Fingers, see *Flexors of fingers*
- Finsen light, vaccinal action, R.S.M. 155, 194
- Finsen's light method in treatment of lupus, Electr. 73, 74, 80
- FINZI, N. S.—Case of sarcoma of ear and mastoid region under radium treatment, Clin. 157
- discussion on case for diagnosis, Clin. 148
- of oesophageal stenosis in a man aged 59, Laryng. 118
- of treatment by radium of malignant growths, Clin. 166
- on examination of intestines by X-rays, Electr. 124
- on treatment of naevi by electricity, cautery and refrigeration, Electr. 48
- on X-ray treatment of lymphadenoma, Electr. 132
- , radium in treatment of malignant growths, Electr. 53; discussion (p. 68): Dr. Sloan, Mr. Deane Butcher, Mr. F. C. Wallis, Mr. Thurstan Holland, Dr. McCulloch, Dr. H. Orton, Dr. Finzi (reply)
- , and HILL, W.—Case of oesophageal growth under treatment by radium, Clin. 160; discussion, (p. 162): Dr. Hill, Dr. Finzi
- , malignant growth in neck treated with radium, with recurrence in mediastinum, Clin. 159
- and SHAW, E. H.—Further report on a case of carcinoma of the breast treated by radium, Clin. 1; discussion (p. 3): Mr. Pearce Gould
- FIRTH, A. C. D.—Prolapse of intestine through ectopia vesicæ, Child. 55; discussion (p. 55): Mr. A. R. Thompson, Dr. Firth (reply)
- and LEDINGHAM, J. C. G.—Hemiatrophy of cerebral hemisphere, Child. 53
- and SPRIGGS, E. I.—Congenital malformation of heart, showing complete atresia of the pulmonary artery, Child. 49; discussion (p. 50): Dr. J. Porter Parkinson
- Fiske Memorial Prize Essay on control of scarlet fever, Epid. 86
- Fistula, biliary, formation of, followed by acute emaciation, treated by cholecyst-duodenostomy (J. D. Malcolm), Clin. 83
- FITZWILLIAMS, DUNCAN C. L.—Discussion on diagnosis and treatment of intussusception in children, Child. 174

- Flatulence (intestinal), treatment by application of lactic-acid bacillus, Therap. 62
- FLEMING, ALEXANDER.—Discussion on vaccine therapy, R.S.M. 137
- , discussion on present position and treatment of syphilis, Surg. 219
- , pathology of acne, quoted, Path. 172, 174
- FLETCHER, H. MORLEY.—Discussion on case of blue patches, Child. 134
- , discussion on case of clicking noise in left ear, Clin. 94
- , on cases of oxycephaly, Child. 127
- , on thoracic aneurysm in boy, Clin. 36
- , "rheumatic nodules" in case of (probably) gonococcal arthritis, Clin. 107; discussion (p. 108): Dr. Poynton, Dr. Garrod, F.R.S., Dr. Morley Fletcher (reply)
- , symmetrical trophic lesions of the extremities in a child, syringomyelia (Morvan's type), Clin. 98; discussion (p. 103): Dr. Henry Head, F.R.S., Dr. Lakin, Dr. Fletcher (reply)
- Flexner, anti-serum treatment of cerebrospinal meningitis, quoted, R.S.M. 85, 86
- Flexors of fingers, spasm of (G. A. Sutherland), Clin. 19
- Flies in milk, spread of diarrhoea by, Epid. 129
- , transmission of diarrhoeal infection by, Epid. 97, 127
- , see *House-flies*
- Flushing, facial, treatment by application of vacuum electrode (static electricity), Electr. 82
- Fœtus, differentiation between male and female pelvis in, Path. 110
- Fog, absent in Naples, Baln. 18
- Folk-medicine, value of, in therapeutics, Therap. 3, 4
- Follicular wall, Odont. 158
- Food, mastication, importance of, Odont. 2, 3
- , pain in duodenal ulcer relieved by, Surg. 70
- , quality of, effect on teeth, Odont. 7
- , value of lactic-acid bacillus as, Therap. 61
- , with preservatives added, avoidance in chronic gastric ulcer, Therap. 132
- Food and Drugs Act, suggestion to make applicable to proprietary medicines, Therap. 100
- Food-stuffs, action of lactic acid on, Med. 3
- , fermentation of, relation to dental caries, Odont. 5
- , oxalates of, derivation of calcium oxalate from, Med. 4
- Foot, tuberculous disease of, treatment by partial tarsectomy (C. P. Childe), Child. 158
- Forceps (high), delivery by, when dangerous, Obst. 128
- (vulsellum-catch) for securing submerged tonsils (H. Tilley), Laryng. 124
- Ford Bank experiment in stamping out bovine tuberculosis, Epid. 240
- FORDYCE, A. D.—Cases of subacute liver atrophy, persistent vomiting and lymphosarcoma, with notes on liver pathology in childhood, Child. 1; discussion (p. 13): Dr. F. Parkes Weber
- Foreign bodies in lung (H. J. Davis), Laryng. 75
- in œsophagus (H. J. Davis), Laryng. 75, 76
- , extraction or dislodgment, technique of, Laryng. 76, 77
- body, soft, in bronchus (D. R. Paterson), Laryng. 24
- Forel, neurone-concept due to, Path. 1
- , on abstinence from salt, quoted, Baln. 62
- Forssman, experiments showing chemiotropic action of degenerated myelin on nerve-fibres, Path. 26
- FORSYTH, DAVID.—Case of congenital œdema with dilatation of the intestinal lymphatics, Child. 51
- , case of non-cretinous mental deficiency with goitre, Child. 95
- , discussion on case of enlarged spleen, Child. 56
- , of myelogenous leukaemia in infant, Child. 94
- Fossa (left nasal), floor of, and maxillary antrum, traumatic (post-operative) perforation through hard palate communicating with (L. H. Pegler), Laryng. 146
- (posterior), two cases of cholesteatoma of unusual size extending into, and causing obliteration of, sigmoid sinus (G. Wilkinson), Otol. 82

- Foul mouth, frequency and danger of, Odont. 97-102
- Foulerton, A. G. R., and Kellas, bactericidal agency of nitric acid and ozone formed in brush discharges, Electr. 84
- Fournier d'Abbe, "Election Theory," quoted, Electr. 74
- Fowler, F.—Discussion on relative value of various transformers, Electr. 159
- FOWLER, J. K.—Discussion on vaccine therapy, R.S.M. 107
- , incipient pulmonary tuberculosis, quoted, Therap. 25
- , position of bacteriologists, quoted, R.S.M. 78
- Fowler position, value of, in operations, Obst. 9
- Fowls, intra-abdominal grafting of testicular tissue in, attempts at, Path. 132, 134, 135
- FOX, H. CLAYTON.—Discussion on bilateral abductor paralysis, Laryng. 162
- , discussion on case of asthma, improvement after nasal treatment, Laryng. 117
- , on case of atresia of the anterior naris, Laryng. 115
- , on cast of upper jaw from case of congenital occlusion of posterior naris, Laryng. 115
- , on perforation of nasal septum from salt dust, Laryng. 5
- , on treatment of deflection of triangular cartilage by septal fissure, Laryng. 129
- , —, — of tuberculosis of larynx, Laryng. 153
- , —, — on tuberculosis of larynx in girl, Laryng. 127
- , —, — on tumour of post-nasal space, Laryng. 161
- , —, — on vulsellum-catch forceps, Laryng. 125
- , paralysis of right vocal cord in a case of myotonia atrophica, Laryng. 20; discussion (p. 20): Dr. Batten, Dr. Grant, Mr. Fox (reply)
- FOX, R. FORTESCUE.—Discussion on arthritis deformans, Baln. 95
- FOX, T. COLCOTT.—Case of dermatitis artefacta, Derm. 28; discussion (p. 29): Dr. Sequeira, Dr. Pernet
- , case of granuloma annulare, Derm. 104
- , discussion on case of acroteric scleroderma, Derm. 34
- , —, — on case for diagnosis, Derm. 14, 18
- , —, — of disseminated lupus in girl aged 4½ years, Derm. 59
- , —, — of epidermolysis bullosa, Derm. 74
- , —, — of k  ratodermie blennorrhagique, Derm. 80
- , —, — ? mycosis fungoides, Derm. 64
- , —, — of pseudo-xanthoma elasticum, Derm. 70
- , —, — of xeroderma pigmentosa with unusual features, Derm. 53
- , —, — on curious case of affection of finger- and toe-nails, Derm. 7
- , —, — on linear n  vus of unusual type (n  vus acneiformis), Derm. 70
- , —, — on lupus erythematosus, Derm. 18
- , —, — on unusual condition of lips, Derm. 19
- , see also under *Excoriations (neurotic) of "dug-out" type*
- FOX, W., and MORRIS, Sir M.—Case of lupus erythematosus, Derm. 99
- Fox, Wilson, inhalation treatment of consumption, quoted, Therap. 19
- Fracture, extracapsular, of neck of femur in boy (A. R. Thompson), Child. 22
- , present in case of cyst of humerus (H. Lett), Child. 98
- , transverse, of both patell   with much separation of fragments, treated by operation (F. S. Eve), Clin. 78
- , of base of skull; operation; recovery (C. A. Ballance), Clin. 62
- , of humerus at site of innocent cyst (R. C. Elmslie), Clin. 192
- , of hyoid bone ("garrotter's throat") (H. J. Davis), Laryng. 77
- , of nasal septum complicating maxillary suppuration; operations; recovery (J. Donelan), Laryng. 89
- , dislocation of spine in mid-cervical region resulting in transverse lesion in lower cervical region of cord (S. V. Sewell and H. H. Turnbull), Path. 61
- , (vertical) through mandible and canine tooth (W. de C. Prideaux), Odont. 92
- Fractures, unobserved, in early life, causing shortening of right humerus and left thigh (H. Burrows), Child. 165

- Franzensbad, cure of female sterility at, explained, *Baln.* 10
- Fraser, results of lesions in posterior longitudinal bundle of animals, quoted, *Path.* 70
- FREEMAN, JOHN.—Discussion on vaccine therapy, *R.S.M.* 97
- FRENCH, Major H. C., R.A.M.C.—Discussion on present position and treatment of syphilis, *Surg.* 222
- FRENCH, HERBERT S.—Bronzed diabetes, with cirrhosis of both liver and pancreas, and pigmentary deposits in these organs as well as in the skin, *Path.* 94
- , case of extensive, yet incomplete, fibro-caseous disease of both suprarenal capsules in which symptoms of Addison's disease were not present, *Path.* 93
- , discussion on treatment of gastric ulcer, *Therap.* 135
- Friction in pneumococcal pericarditis, how far present, *Med.* 89
- sounds absent in purulent pericarditis, *Med.* 72
- — at base in pericardial effusion, *Med.* 59, 60
- — in pericarditis, preceded by compression of lower lobe of left lung, *Med.* 83, 84
- Friedmann, tuberculous infection of tonsils in children, quoted, *Child.* 30
- Frog-child (Batten's) (T. H. Openshaw), *Clin.* 39
- Frogs, experiments on, to test truth of neurone theory, *Path.* 6-8
- Frontal bone, removal of, for septic osteomyelitis (C. J. Symonds), *Laryng.* 40
- Frontal sinus, disease of, with necrosis (W. H. Kelson), *Laryng.* 28
- — —, treatment by vaccine, *R.S.M.* 189
- — —, extension of tumour to, after excision of both superior maxillæ (A. Carless), *Clin.* 114
- — — (radical operation), two cases showing conditions nine and sixteen months after operation (StClair Thomson), *Laryng.* 144
- — — (left), chronic suppuration of, with displacement of the left eyeball and diplopia; operation: recovery (C. Nourse), *Laryng.* 110
- Fruit, eating of, in causation of summer diarrhœa, *Epid.* 139
- "Fulguration" in treatment of malignant disease, *Electr.* 40
- Furunculosis, associated with pruritus ani, treatment by vaccine, *R.S.M.* 15
- GABELL, D. P.—Discussion on case of painful attrition, *Odont.* 120
- , discussion on odontoceles and some other cysts, *Odont.* 160
- — — on spindles of von Ebner, *Odont.* 150
- — — on susceptibility of teeth to dental caries, *Odont.* 81
- — — on unerupted teeth and film radiographs, *Odont.* 89
- GADD, H. WIPPELL.—Report on a drug known as "kangalugi," *Therap.* 67
- Gaertner's bacillus and summer diarrhœa, *Epid.* 133
- GALLOWAY, JAMES.—Discussion on case of congenital hairy mole, *Clin.* 198
- , discussion on case of jaundice with unpigmented skin, *Clin.* 197
- — — on cases of adiposis dolorosa, *Clin.* 60
- — — — of angioneurosis, *Clin.* 106
- — — on case of pityriasis rubra, *Derm.* 61
- — — on condition of mouth in chronic disease, *Odont.* 107
- — — on bluish pigmentation of the skin, *Clin.* 141
- — — on scleroderma and sclerodactyly, *Clin.* 34
- , spleno-medullary leukemia: intercurrent erysipelas, *Clin.* 135; discussion (p. 138): Dr. H. D. Rolleston, Dr. F. Parkes Weber, Dr. Galloway (reply)
- , "trophic" dermatitis commencing on the extremities, *Clin.* 139
- and WILLIAMS, A. W.—Lichen planus in a diabetic, *Derm.* 45; discussion (p. 46): Dr. Galloway, Dr. Adamson, Dr. Whitfield, Dr. Williams (reply)
- Gall-stones, appendicular gastralgia diagnosed as, *Surg.* 203
- (cholesterol), containing typhoid bacilli (A. F. Hertz and F. S. Adams), *Clin.* 169
- , diagnosis from duodenal ulcer, *Surg.* 88, 89, 90, 92
- , gastric hypersecretion associated with, *Surg.* 181, 182, 184, 185

- Galvanic current, technique in gynecological applications, Electr. 33
- Galvanism in treatment of sclerodermia and sclerodactyly, Clin. 32
- Galvano-cautery and sanatorium treatment healing tuberculosis of epiglottis, left ary-epiglottic fold, and left inter-arytæoid space (StC. Thomson), Laryng. 7
- , in treatment of lupus of epiglottis, Laryng. 87
- of nævi, Electr. 39, 46
- of tuberculosis of larynx, almost complete recovery resulting (J. Dundas Grant), Laryng. 87
- , seven applications of, cicatrizing tuberculosis of vocal cords (StC. Thomson), Laryng. 6
- Galvano-puncture, in case of healed tuberculous laryngitis, (H. Tilley), Laryng. 121
- GAMBLE, F. W.—Discussion on proprietary, patent and secret remedies, Therap. 93
- Ganglion-cells, chromatic changes in, after cobra-venom poisoning, Neur. 110, 111
- Gangrene, result of rupture in hydrocele, Surg. 167
- GARDNER, H. BELLAMY.—Lymphatism, with pathological report by R. Salusbury Trevor, Anæsth. 19-36; discussion (p. 37): Dr. Dudley Buxton, Dr. Spilsbury, Dr. Hewitt, Dr. Horace Manders, Dr. Leonard Williams, Mr. Carter Braine, Dr. Farquhar Buzzard, Dr. Blumfeld, Dr. Ralph Vincent, Dr. A. G. Levy, Mr. R. E. Humphry, Dr. A. S. Currie, Mr. Bellamy Gardner (reply)
- GARDNER, T. F.—Discussions on arthritis deformans, Baln. 96
- GARROD, A. E.—Discussion on arthritis deformans, Baln. 99
- , discussion on case of achondroplasia in twin, Child. 43
- on Meckel's diverticulum containing calculi, Clin. 15
- on rheumatoid arthritis with enlargement of lymphatic glands and spleen, Clin. 18
- on "rheumatic nodules" in probable gonococcal arthritis, Clin. 108
- multiple peripheral neuritis in a child, Child. 38; discussion (p. 39); Dr. Garrod, Dr. G. A. Sutherland, Dr. E. I. Spriggs, Dr. Garrod (reply)
- "Garrotter's throat" (fracture of hyoid bone) (H. J. Davis), Laryng. 77
- Gartner's duct, apparent development from, of cystic tumour of right broad ligament, springing from uterus (A. H. N. Lewers), Obst. 67
- Gasserian ganglion, persistence of neuralgic pain after removal of, Neur. 87
- Gastou, Dr., on the ultra-microscope, quoted, Clin. 6
- "Gastralgia, appendicular," correctly diagnosed, Surg. 188
- , or the appendix as cause of gastric symptoms (H. J. Paterson), Surg. 187
- , sex incidence, Surg. 196, 204, 205, 207
- , symptoms, Surg. 194
- , hæmorrhagic, Surg. 205
- Gastrectasia, Electr. 113
- Gastrectomy by Billroth's first method, Surg. 140, 141
- by Billroth's second method, Surg. 140, 141
- by Kocher's method, Surg. 140
- for supposed cancer of stomach, Surg. 138
- performance in two stages, Surg. 158
- Gastric crises, differential diagnosis from, Surg. 107
- disturbances in phthisis, connexion with pyorrhœa alveolaris simultaneously present, R.S.M. 17
- hypersecretion and connexion with latent disease of appendix (W. S. Fenwick), Surg. 177
- juice, hyperacid action of, in production of gastric ulcer, Therap. 124, 126, 132
- stasis in duodenal ulcer, Surg. 73
- ulcer, rupture of, during dental operation (W. de C. Prideaux), Odont. 91
- Gastritis, appendicitis following, Surg. 202
- , causes of, Surg. 181
- , fats in treatment of, Med. 33, 34
- , hæmatemesis in, Surg. 195

- Gastritis (phlegmonous), from hydrochloric-acid poisoning; vomiting of complete mucous membrane of pyloric half of stomach; recovery (S. A. Clarke, A. F. Hertz, and R. P. Rowlands), Clin. 209
- Gastrocnemius muscle, position of attachment, Child. 57, 58
- Gastro-enterostomy, advances in, Surg. 117
- as preliminary operation for cancer of stomach, Surg. 142-144, 147
- for supposed cancer of stomach, Surg. 138, 139
- in treatment of chronic gastric ulcer, Therap. 129, 131
- — duodenal ulcer, Surg. 78, 79, 81, 92, 93, 95
- — — when contra-indicated, Surg. 115, 116
- , posterior, first step in operation for removal of lymphatic area connected with stomach, Surg. 152
- , useless in hæmorrhage in acute gastric ulcer, Therap. 124
- Gastro-jejunostomy, avoidance of, by removal of appendix, Surg. 181
- , contra-indications for, Surg. 207, 208
- , disappearance of gastric hypersecretion after, Surg. 182
- , gastric symptoms not cured by, Surg. 202, 203, 208
- in treatment of chronic gastric ulcer, Therap. 129
- of duodenal ulcer, Surg. 109, 110
- , preliminary to operation for cancer of stomach, Surg. 159
- , rules for employment of, Surg. 203, 208
- Gastropsis, diagnosed by X-rays, Electr. 113
- and incipient disease of appendix, Surg. 184
- Gastrostaxis, Surg. 195, 205
- , synonymous clinically with acute gastric ulcer, Therap. 136
- "Gastrosuccorrhœa," Surg. 177
- Gelatine treatment (Senator's method) of chronic gastric ulcer, Therap. 131
- General Medical Council, Pharmacopœia Committee, resolution dealing with British Pharmaceutical Codex, Therap. 93, 97
- — —, relations with Pharmaceutical Society, Therap. 94, 97
- General practice, infections most commonly met with in, R.S.M. 103
- — —, vaccine therapy in, R.S.M. 102
- General practitioners, demands made on knowledge by patients, R.S.M. 64, 65
- — — and consultants, responsibility for correct diagnosis and treatment of cases falls jointly upon, R.S.M. 6
- Generative organs of Chinese eunuch, Path. 103, 124-126
- — —, female, diseases of, incipient stages often more painful than later, Obst. 4
- — —, serious conditions often unaccompanied by much pain, Obst. 4, 5
- Gengou and Bordet, on complement-deviation, quoted, R.S.M. 163
- Genital organs (female), pain associated with (H. Macnaughton-Jones), Obst. 1
- Genitals (male, external), complete ablation, specimen, Path. 103
- Genito-urinary diseases, treatment by vaccines, R.S.M. 63
- — tract, tuberculosis of, treatment with vaccine, results, R.S.M. 147
- Gentile, stomach evacuator, Surg. 178
- Germany, geographical distribution of hyperchlorhydria in, Surg. 101
- , obstetric clinics of, puerperal morbidity in, Obst. 222
- Gerster, A. G., letters to Dr. Coley on results of treatment of inoperable sarcoma by mixed toxins, Surg. 47, 48
- Gestation, ectopic, specimen of (S. J. Aarons), Obst. 130
- Geyer, A. C., nævi treated by refrigeration, Electr. 41
- Giantism, acromegalic, case of (R. Milne), Clin. 54
- Gibbon, Captain, R.A.M.C., Wassermann serum diagnosis of syphilis, Surg. 223, 224
- Gilchrist, pathology of acne, Path. 174
- GILES, A. E.—Case of kraurosis of the vulva, with commencing carcinoma, Obst. 54
- , discussion on leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, Obst. 48

- GILES, A. E.—Discussion on pelvic hæmatocele of ovarian origin, *Obst.* 111
 —, large solid ovarian tumour: adeno-carcinoma? *Obst.* 51; discussion (p. 52): Dr. H. Spencer, Dr. A. Routh, Dr. Lockyer, Dr. Briggs, Dr. Macnaughton-Jones, Dr. Glendining, Dr. Darwall Smith; report of Pathological Committee on specimen
 —, very large cervix fibroid, *Obst.* 184; discussion (p. 185): Dr. Purslow, Dr. Macnaughton-Jones, Dr. Florence Willey
- GILL, R.—Discussion on apparatus for administration of ether by purely open method, *Anæsth.* 18
 —, discussion on choice of anæsthetic, *Anæsth.* 73
 —, on heart massage in heart failure during anæsthesia, *Anæsth.* 10
- Glanders, chronic, in a man (G. S. Hett), *Laryng.* 1
 —, difficulties of formation of antibodies in, *R.S.M.* 66, 67
 —, relation of temperature to opsonic index in case of, *R.S.M.* 42, 43
 —, treatment by vaccine, *R.S.M.* 66
- Glands (cervical), slight enlargement in case of endolaryngeal cancer (W. Hill), *Laryng.* 168
 —, enlarged, disappearance of, in lymphadenoma under treatment by X-rays (A. H. Pirie), *Electr.* 125-131
 —, in mesentery, chylous cyst of mesentery, associated with (H. Burrows), *Child.* 164
 —, internal accessory, transmission of stimulation to, by skin, *Baln.* 4, 5, 7
 —, secretory, cutaneous stimulation of, *Baln.* 4, 5, 7
 —, disorders of, characterized by abnormalities of dermic pigmentation, *Baln.* 5
 —, effects of climate on, *Baln.* 5
 —, physiological antagonism, *Baln.* 11
 — (tuberculous), non-suppurating, treatment with tuberculin, *R.S.M.* 105
 —, treatment with vaccine, results, *R.S.M.* 147
 — (cervical, tuberculous), case illustrating value of opsonic index, *R.S.M.* 45
 —, not always afebrile, *R.S.M.* 212
 —, with secondary staphylococcic infection, case recovering after treatment with tuberculin, *R.S.M.* 115
- Gland-shadows, diagnosis of ureteral calculus from, *Electr.* 94
- Glasgow Maternity Hospital, statistics of puerperal eclampsia from, *Obst.* 201
- GLENDINING, B.—Discussion on fibro-adenoma of ovary, *Obst.* 53
 —, discussion on malignant and innocent ovarian growths, *Obst.* 96
 — and PARSONS, J. INGLIS.—Adenomyoma of the Fallopian tube, with tuberculous salpingitis, *Obst.* 238; report of Pathology Committee (p. 239); discussion (p. 240): Dr. Macnaughton-Jones, Dr. Glendining (reply)
- Glia cells, changes in, after snake-venom poisoning, *Neur.* 111
- Glioma of brain and epilepsy, *Neur.* 2, 4, 10
 — of pineal body, case quoted, *Neur.* 70, 71
- Glottis, laryngo-fissure for sub-glottic enchondroma (StC. Thomson), *Laryng.* 13
- Glycosuria, choice of anæsthetic for subjects of, *Anæsth.* 70
 —, treatment by vaccine, *R.S.M.* 18, 178-181
- GOADBY, K. W.—Bacteriology of pyorrhœa alveolaris, *Odont.* 30, 31, 32, 33, 61, 67
 —, discussion on vaccine therapy, *R.S.M.* 86
 —, vaccine therapy in pyorrhœa alveolaris, quoted, *R.S.M.* 62
 —, treatment of early cases of pyorrhœa alveolaris, *Odont.* 55
- Godard on growth of eunuchs, *Path.* 106
- GODLEE, R. J.—Discussion on radical operation for cancer of pylorus, *Surg.* 158, 163
 — on rupture of tunica vaginalis in hydroceles, *Surg.* 177
 —, osteo-arthritis and trauma, quoted, *Baln.* 90
 —, presidential address: prognosis in certain inflammatory diseases of the lungs and pleura commonly treated surgically, *Surg.* 49-68
- GOFFE, E. G. L.—Discussion on treatment of nævi by electricity, cautery and refrigeration, *Electr.* 50
- Goitre (exophthalmic), case occurring in boy (E. Pritchard and S. Stephenson), *Child.* 73

- Goitre (exophthalmic) in man with symmetrical telangiectases of the ocular conjunctivæ (F. Parkes Weber), Clin. 142
- , with lymphatism, case operated for under ether, Anaesth. 49
- , non-cretinous mental deficiency with (D. Forsyth), Child. 95
- , prevalence of, in Chilterns, Baln. 45
- , specimen of, with portions of larynx and trachea from woman aged 37 (J. Donelan), Laryng. 38
- Goldsschend, results of gastrectomy, quoted, Surg. 140, 141
- Goldthwait, osteo-arthritis, quoted, Baln. 87, 92
- Golgi, chrome-silver method of, in demonstration of anatomy of nervous system, Path. 3, 4
- Gonococcus, opsonic index, R.S.M. 121
- , see also *Arthritis (gonococcal)*; *Iritis (gonococcal)*
- Gonorrhœa, followed by acute prostatitis, Electr. 148
- , relation of certain cases to true rheumatism, Clin. 108
- , treatment by vaccine, R.S.M. 104
- , — by vacuum electrode, Electr. 148
- GOODALL, E. W.—Discussion on control of scarlet fever, Epid. 87
- GOODBODY, F. W.—Discussion on therapeutic value of lactic-acid bacillus, Therap. 56
- GOODHART, J. F.—Discussion on measles and its pre-eruptive symptoms, Epid. 65
- , discussion on scarlet fever, its home treatment and prevention, Epid. 65
- , on future of cancer research, quoted, Therap. 74
- Goodman, on pancreatic reaction, quoted, Med. 181
- Gordon, M. H., on *Streptococcus pyogenes*, quoted, Otol. 20
- GOSSAGE, A. M.—Discussion on, and exhibition of, cases of adiposis dolorosa, Clin. 58
- , discussion on dorsal percussion of thorax and stomach, Med. 240
- , and MARRIS, H. F.—Statistics of pericardial effusion from Westminster Hospital, Med. 110
- GOULD, A. PEARCE.—Detachment of entire scalp, Clin. 129
- , discussion on aneurysm of descending thoracic aorta, Clin. 128
- , — on case of bending of bones of both legs and partial ankylosis of both hips, Clin. 95
- , — of cancer treated by radium (further report), Clin. 3
- , — of excision of both maxillæ, Clin. 11
- , — of fracture of humerus at site of innocent cyst, Clin. 195
- , — of impaction of large calculus in lower end of right ureter, Clin. 65
- , — of jaundice with areas of unpigmented skin, Clin. 198
- , — on cases of adiposis dolorosa, Clin. 58
- , — on cholecyst-duodenostomy for acute emaciation following formation of biliary fistula, Clin. 85
- , congenital treatment in left parotid region, Clin. 144
- , — on enlargement of tubercle of tibia, Clin. 191
- , — on extensive myositis ossificans, Clin. 154
- , — on Meckel's diverticulum containing calculi, Clin. 15
- , — on popliteal aneurysm in both legs, Clin. 132
- , — on rheumatic spondylitis with torticollis and subluxation, Clin. 38
- , — on thoracoplasty in treatment of empyema, Clin. 91
- , — on treatment of stricture of rectum and other conditions by fibrolysin, Clin. 89, 90
- , removal of vesical calculus weighing 8 oz., Clin. 31
- , tribute to the memory of the late Mr. H. H. Clutton, Clin. 29
- Gout, importance of examination of teeth in cases of, Odont. 95
- , in relation to arthritis, Baln. 92
- , question of giving or withholding saline waters in cases of, Baln. 78, 79, 81, 84
- , rarity in Ireland, Clin. 24
- , — among Mahommedans, Clin. 22
- , with tophaceous deposits in Mahommedan (Sir D. Duckworth), Clin. 21
- , with uratic tophaceous deposits in boy aged 14 (Sir D. Duckworth), Clin. 23

- Gow, A. E., and BROWN, W. LANGDON.—Statistics of pericardial effusion from St. Bartholomew's Hospital, Med. 134
- Gow, W. J.—Discussion on Cæsarean section in eclampsia gravidarum, Obst. 205
- , discussion on Cæsarean section in placenta prævia, Obst. 179
- GOWERS, Sir W., F.R.S.—Criticism of view of meningitis being associated with early phenomena of syphilis, Neur. 36
- , special sense discharges from organic disease (Hughlings Jackson Lecture), Neur. 1
- Gowring, W. B., rupture of gastric ulcer during dental operation, quoted, Odont. 91
- Grafting, experiments: see *Bones (fætal)*; *Does (pregnant)*; *Rabbits (testicle)*; *Thymus gland*
- Gram's method, examination of cover-glass preparations of fæces by, Therap. 56
- GRANT, J. DUNDAS.—Case of endothelioma of the temporal bone, Otol. 32; discussion (p. 33); Dr. P. McBride, Mr. A. Cheate, Dr. D. R. Paterson, Mr. C. H. Fagge, Dr. Grant (reply)
- , case of malformation of soft palate and uvula in girl aged 16, Laryng. 131; discussion (p. 131): Dr. Donelan, Dr. Jobson Horne
- , of probable late secondary specific pharyngitis with nerve symptoms, Laryng. 154; discussion (p. 154): Dr. F. Powell, Mr. A. L. Whitehead, Dr. Donelan, Dr. Grant (reply)
- , of removal of the hypertrophied anterior lip of the hiatus semilunaris for long-standing catarrh, Laryng. 131; discussion (p. 132): Mr. H. Tilley, Dr. Grant (reply)
- , destructive ulceration of the hard and soft palate in a young male adult, with exfoliation of large mesial sequestrum, apparently of specific origin, in a tuberculous subject, Laryng. 71
- , discussion on bands between Eustachian tube and pharynx, Laryng. 69
- , on case of asthma: improvement after nasal treatment, Laryng. 117
- , of audible tinnitus, Otol. 55
- , of benign laryngeal growth, Laryng. 94
- , of "bleeding polypus" of inferior turbinate, Laryng. 106
- , of chronic laryngitis with prolapse of ventricle on right side, Laryng. 30, 31
- , for diagnosis, Laryng. 92, 140
- , of general infiltration of larynx, probably luetic, Laryng. 71
- , of hoarseness in woman, Laryng. 56
- , of one month's duration in a woman aged 60, Laryng. 31
- , hypertrophic laryngitis with stenosis in syphilitic subject, Laryng. 93
- , on Kuhn's instruments for per-oral intubation, Otol. 50
- , on case of large chronic swelling in tonsillar region, Laryng. 133
- , of laryngeal new growth for diagnosis, Laryng. 60, 62
- , paralysis, Laryng. 151
- , of leukoplakia of soft palate, Laryng. 83
- , of paresis of palate, pharynx, and œsophagus, Laryng. 91
- , of pemphigus vegetans affecting larynx and oral cavity, Laryng. 78
- , of "singer's node" on left cord, Laryng. 108
- , of specific ulceration of lower lip, inside of the cheek and tongue, Laryng. 69
- , on congenital absence of bony part of palate, Laryng. 163
- , on deformity of both pinnae, Otol. 72
- , on diseases of the ventricle of the larynx, Laryng. 159
- , on epithelioma of larynx, Laryng. 141
- , on excision of entire tongue, Laryng. 138-139
- , on large nasopharyngeal growth in boy aged 12, Otol. 48
- , on laryngeal growth, probably malignant, Laryng. 74
- , vertigo, Laryng. 81
- , on left abductor paralysis, Laryng. 137
- , on left facial paralysis following mild catarrh of middle ear, Otol. 60
- , on loss of voice with dyspnoea, Laryng. 163
- , on model demonstrating "back" and "belly" breathing, Laryng. 49, 50

- GRANT, J. DUNDAS.—Discussion on objective clicking sound in left ear, Otol. 37
- , discussion on obstruction of both nostrils, Laryng. 149
 - on papillomata of larynx from boy aged 6, removed by direct method, Laryng. 98
 - on paralysis of right vocal cord in myotonia atrophica, Laryng. 21
 - on party-wall pharyngeal cancer, Laryng. 143
 - on pathology of cholesteatomata, Otol. 68
 - on pedunculated papilliform growth springing from posterior border of cartilaginous meatus, Otol. 46
 - on perforation of nasal septum from salt (NaCl) dust, Laryng. 4, 5
 - on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 58
 - on radical frontal sinus operation, Laryng. 145
 - on rare congenital deformity of nose, Laryng. 85
 - on removal of penny from gullet, Laryng. 136
 - of a rapidly growing soft fibroma from posterior wall of left maxillary antrum by modified Rouge's operation, Laryng. 97
 - on sanatorium, silence and galvano-cautery treatment of tuberculosis of throat, Laryng. 10
 - on specimen of larynx showing re-development of epithelioma on left cord after removal of right for same cause, September, 1896, Laryng. 34
 - on swelling on left side of neck accompanied by dyspnoea, Laryng. 22
 - on thyroid gland operation in extra-laryngeal inoperable carcinoma, Laryng. 16
 - on treatment of deflection of triangular cartilage by septal fissure, Laryng. 130
 - of tuberculous laryngitis by galvano-puncture, Laryng. 123
 - on tuberculosis of larynx, Laryng. 164
 - of larynx in girl aged 12, Laryng. 128
 - on tuberculous ulcer on right side of tongue, Laryng. 82
 - on tumour of post-nasal space, Laryng. 161
 - on two cases of hoarseness, Laryng. 41
 - on ulcer of doubtful nature on left arytaenoid cartilage, Laryng. 30
 - on ulceration of epiglottis, probably epitheliomatous, Laryng. 145
 - on vulsellum catch forceps, Laryng. 126
 - on well-defined Ménière's symptoms, Otol. 52
 - , epithelioma of right vocal cord in man aged 60, removal by thyrotomy, Laryng. 17; discussion (p. 18): Mr. de Santi, Dr. Scanes Spicer, Dr. Grant (reply)
 - , —, —, seven months after operation; no recurrence; development of cicatricial substitute for vocal cord, Laryng. 156
 - , extensive tuberculosis of the larynx in a middle-aged man with rapid evolution, showing almost complete recovery, galvano-caustic treatment, Laryng. 87; discussion (p. 89): Dr. FitzGerald Powell, Dr. Grant (reply)
 - , growth in the nasopharynx of an elderly female, like very pale "adenoids," but much more dense in consistence, suggestive of malignancy, Laryng. 72; discussion (p. 73): Mr. Herbert Tilley, Dr. Scanes Spicer, Dr. Grant (reply)
 - , hypertrophy of the lingual tonsil, with impairment of singing voice, improved by a snaring operation, Laryng. 155; discussion (p. 156): Dr. S. Spicer, Mr. Horsford, Dr. Grant (reply)
 - , infiltration of left ventricular band. Neoplasm? Tuberculosis? Laryng. 16
 - of the right half of the larynx of obscure nature in a woman aged 34, Laryng. 146; discussion (p. 147): Dr. Watson Williams
 - , microscopical section from fragment of an adenoid-like growth in the nasopharynx of a middle-aged lady, probably an early stage of epithelioma, Laryng. 73
 - , odynphagia in a tuberculous subject, without any obvious disease of the larynx, Laryng. 147
 - , peculiar erythema of the left palate in a man aged 41 who had been a considerable smoker, Laryng. 27; discussion (p. 27): Mr. Barwell, Dr. H. J. Davis, Dr. Dan McKenzie, Dr. Grant (reply)

- GRANT, J. DUNDAS.—Secondary specific pharyngitis in a young woman, *Laryng.* 28; discussion (p. 28): Dr. Donelan
- , tuberculosis of the larynx, with extreme odynphagia relieved by injection of alcohol into the left superior laryngeal nerve, *Laryng.* 163; discussion (p. 153): Mr. M. Hovell, Dr. Watson Williams, Dr. Donelan, Mr. Howarth, Mr. C. Fox, Dr. Grant (reply)
- and MCKENZIE, D.—Epithelioma of left vocal cord, in woman aged 58, removal by thyrotomy, *Laryng.* 19
- Granuloma annulare, case of (H. Davis), *Derm.* 57
- — — (T. C. Fox), *Derm.* 104
- Graves' disease, see *Goitre (exophthalmic)*
- Graves, seasonal prevalence of scarlet fever in Ireland in early nineteenth century, quoted, *Epid.* 75, 88, 93
- GRAY, A. A.—Lantern-slide demonstration, illustrating the pathological changes found in the ear of deaf mutes, *Otol.* 11; discussion (p. 13): Mr. Yearsley, Dr. Urban Pritchard, Mr. S. Scott, Mr. C. G. Lee, Dr. Gray (reply)
- Gray, H. Tyrrell, application of spinal anæsthesia in irreducible intussusception of children, *Child.* 175
- Great Britain, cycle of prosperity in (1871-80), *Epid.* 10, 11
- — — — —, beneficial effects on public health, *Epid.* 10, 11
- GREEN, S.—Discussion on laryngo-fissure for subglottic enchondroma, *Laryng.* 13
- GREENWOOD, M. jun.—Discussion on bovine tuberculous mastitis, *Epid.* 245
- , discussion on communication of diarrhoea from sick to healthy, *Epid.* 126
- Grey oil, intramuscular injections of, in syphilis, *Surg.* 213, 235, 236
- GRIFFITH, W. S. A.—Discussion on Cæsarean section in placenta prævia, *Obst.* 13, 180
- , discussion on double uterus with dysmenorrhœa, *Obst.* 242
- — — on malignant and innocent ovarian growths, *Obst.* 97
- — — on modern methods of delivery in contracted pelvis, *Obst.* 126
- , large cervical fibroid in the vagina obstructing labour, *Obst.* 188
- , specimen of torsion of the body of uterus, *Obst.* 187; discussion (p. 188): Dr. H. Macnaughton-Jones
- Grocco's area of triangular dullness, *Med.* 218, 227
- — — — — value in diagnosis, *Med.* 239, 240
- sign, *Clin.* 27, *Med.* 214, 217
- — —, range of, *Med.* 219
- — —, test and counter-test, *Med.* 218
- GROEDEL, F. M.—Examination of the intestines by means of Röntgen rays, both under normal and pathological conditions, *Electr.* 109; discussion (pp. 120-124): Dr. Herschell, Dr. Hertz, Mr. Holland, Dr. Pirie, Dr. H. Orton, Dr. C. Wilson, Dr. W. Ewart, Dr. Barclay, Dr. C. J. Morton, Dr. Finzi, Dr. Groedel (reply)
- Gross's test for pancreatic insufficiency, *Med.* 201
- GROVES, E. W. HEY.—Specimen of tubal mole, *Obst.* 134
- , the radical operation for cancer of the pylorus, with especial reference to the advantages of the two-stage operation, and to the question of the removal of the associated lymphatics, *Surg.* 117-158; discussion (p. 158): Mr. R. J. Godlee, Mr. Paterson, Mr. W. G. Spencer, Mr. Jonathan Hutchinson, Mr. C. A. Ballance, Mr. Groves (reply)
- Growth, new, of right tonsil invading surrounding tissue (A. Evans), *Laryng.* 147
- Gullet of young girl, penny removed from (H. Tilley), *Laryng.* 135
- Gumma of breast simulating malignant disease (H. J. Paterson), *Clin.* 82
- Gynæcology, diagnosis in, "neurosis," "neurasthenia," "hysteria," terms misleading, *Obst.* 3
- , electro-therapeutics in (S. Sloan), *Electr.* 1
- — — — —, origin of, *Electr.* 4
- (medical) reduced to narrow limits, *Electr.* 2, 3
- (operative), want of moderation in, *Electr.* 3

- HACKNEY, J.—Discussion on teaching of therapeutics in hospital wards, *Therap.* 15
- Hæmangioma, capillary (port-wine nævus), association with lymphangioma, *Clin.* 112
- Hæmatemesis, appendicular disease cause of, *Surg.* 191, 197, 207
- , hypersecretion cause of, *Surg.* 182, 185
- in appendicitis, *Surg.* 195, 196
- in duodenal ulcer, *Surg.* 75-77, 80
- in gastric ulcer, *Med.* 119
- in gastritis, *Surg.* 195
- Hæmatocoele, diagnosis of, *Surg.* 166, 172
- , extensive retroperitoneal, complicating large uterine fibroid; operation; cure (W. Alexander), *Obst.* 139
- , localization of, *Surg.* 170
- (pelvic) of ovarian origin, cases of (C. Lockyer), *Obst.* 106; (A. E. Giles), *Obst.* 111
- , puncture of, *Surg.* 173
- Hæmatometra, case of, *Obst.* 242
- Hæmaturia due to presence of calcium-oxalate calculus, *Med.* 8, 9
- Hæmolysins, how produced, *Neur.* 47
- Hæmopericardium, *Med.* 55
- Hæmoptysis, clinical indication of diagnosis of aneurysm of descending thoracic aorta, *Clin.* 126
- Hæmorrhage, cerebral, from luetic vessels (H. Head), *Neur.* 31
- , fatal, in gastric hypersecretion, *Surg.* 180, 185, 186
- from stomach, diagnosis, *Surg.* 96
- in cerebral cortex from case of electric shock, *Path.* 142
- in acute gastric ulcer, treatment, *Therap.* 121, 123
- — — — — operative, *Therap.* 124
- — — — —, dislodgment of clot in, *Therap.* 122, 123
- (occult) in duodenal ulcer, *Surg.* 76, 112
- , symptom of appendicular gastralgia, *Surg.* 195, 202
- treated by appendicostomy in pneumococcal colitis with hyperpyrexia (H. E. Bruce-Porter, J. P. Lockhart Mummery, W. Hale White), *Clin.* 48
- Hæmorrhages (subcutaneous, unusual) in fatal case of poisoning by phosphorus taken as abortifacient (R. G. Hann and R. A. Veale), *Therap.* 64
- Hæmorrhoids, bleeding due to, mistaken for intussusception, *Child.* 177
- in women, treatment by electricity, *Electr.* 7, 12, 14, 19, 27
- Halbertsma, Cesarean section in eclampsia, quoted, *Obst.* 196
- HALL, ARTHUR.—Spasmodic neurosis, ? Paramyoclonus multiplex, *Neur.* 28; discussion (p. 29): Dr. Farquhar Buzzard, Dr. Thomas Buzzard
- HALL, F. DE HAVILLAND.—Discussion on case of trigeminal neuralgia treated by injections of alcohol, *Clin.* 201
- , discussion on case of mycosis fungoides, *Clin.* 8
- — — on laryngeal vertigo, *Laryng.* 80
- HALL, I. WALKER.—Pathological report on malignant growth of the right temporal bone— with extension through the external meatus, resembling an aural polypus, *Otol.* 87
- Hallé, pathology of acne, *Path.* 173
- Halliburton, W. D., F.R.S., researches on pineal gland alluded to, *Neur.* 76
- and Mott, F. W., F.R.S., experiments to determine extent of interference with regeneration of motor nerves, *Path.* 18-20
- HAMER, W. H.—Discussion on control of scarlet fever, *Epid.* 89
- , discussion on summer diarrhoea and enteric fever, *Epid.* 263
- , "Nuisance of flies," alluded to, *Epid.* 154
- Hampden (Great), medical history of, *Baln.* 41
- Hand, beard region, and upper lip, ringworm affecting (S. E. Dore), *Derm.* 111
- Handley, W. Sampson, path of lymphatic permeation, quoted, *Surg.* 147
- HANN, R. G., and VEALE, R. A.—Fatal case of poisoning by phosphorus, taken as an abortifacient, with unusual subcutaneous hæmorrhages, *Therap.* 64

- HADOT and GILBERT, cirrhosis maligna, quoted, *Path.* 150, 151
- HARES (Belgian, male), effects of castration on long bones, pelvis and skull compared with measurements in normal specimen, *Path.* 104, 105
- HARLEY, VAUGHAN.—Discussion on therapeutic value of lactic-acid bacillus, *Therap.* 57
- HARMAN, N. BISHOP.—Abnormal congenital pigmentation of one eye, *Child.* 67
- , two cases of familial discoid or "Coppock" cataract, *Child.* 68; discussion (p. 70): Mr. Sydney Stephenson, Mr. Hartman (reply)
- HARMER, W. D.—Discussion on cases of epithelioma of larynx shown after operation, *Clin.* 119
- , fibrosarcoma of nose removed after temporary ligature of both external carotid arteries and laryngotomy, *Clin.* 122
- HARRIS, A. BUTLER.—Discussion on vaccine therapy, *R.S.M.* 101
- HARRIS, WILFRED.—Case of thrombosis of the left posterior inferior cerebellar artery, followed by severe trigeminal neuralgia in the analgesic facial area, *Neur.* 81; discussion (p. 85): Dr. Hinds Howell, Dr. R. F. Kennedy, Sir Victor Horsley, Dr. Gordon Holmes, Mr. Sydney Scott, Dr. Purves Stewart, Dr. Henry Head, Dr. Campbell Thomson (reply), Dr. Wilfred Harris (reply)
- , chronic meningo-myelitis: internal ophthalmoplegia, *Neur.* 24; discussion (p. 25): Dr. Harris, Dr. Gordon Holmes, Dr. Page May, Dr. Savill, Dr. Harris (reply)
- , discussion on case of tabes with amyotrophy, *Neur.* 28
- , on cases of thrombosis of cerebral arteries and case of cerebral hæmorrhage from luetic vessels, *Neur.* 32
- , on degenerative changes in nervous system, illustrated by snake-venom poisoning, *Neur.* 116
- , on syphilis and parasymphylis of the nervous system, *Neur.* 61
- , myoclonus with spasm of the tongue, *Neur.* 104
- , syringomyelia with total anaesthesia, *Neur.* 101; discussion (p. 103): Professor Petren, Dr. H. R. Prentice, Dr. F. Kennedy, Dr. W. Harris (reply)
- , trigeminal neuralgia of left first and second divisions treated by injections of alcohol, *Clin.* 200; discussion (p. 201): Dr. de Havilland Hall
- , two cases of chronic syphilitic poliomyelitis, *Neur.* 23; for discussion see *Harris, Wilfred*, chronic meningo-myelitis: internal ophthalmoplegia
- and SPILSBURY, BERNARD H.—Acute cerebral softening, ? due to venous thrombosis, *Neur.* 130; discussion (p. 132): Dr. F. E. Batten, Dr. Farquhar Buzzard, Dr. Trevelyan, Dr. Harris (reply)
- HARRISON, E. F.—Discussion on proprietary, patent, and secret remedies, *Therap.* 97
- HARRISON, Captain L. W.—Discussion on present position and treatment of syphilis, *Surg.* 226
- HARRISON, Ross, differential experiment to test neurone theory, *Path.* 6
- , origin and growth of nerve-fibres, *Path.* 10, 11, 12
- Hart, Carl, angiosarcoma of pineal body, case quoted, *Neur.* 70
- HASLIP, G. E.—Discussion on saline waters and common salt, *Baln.* 84
- Hassall, inhalation treatment of consumption, *Therap.* 19
- Hassall's corpuscles, degenerative changes in cases of lymphatism, *Therap.* 40
- HASTINGS, SOMERVILLE.—"Bleeding polypus" of the inferior turbinate, *Laryng.* 102; discussion (pp. 105-106): Mr. F. A. Rose, Dr. J. Dundas Grant, Dr. Pegler, Mr. Hastings (reply)
- , case of bending of the bones of both legs and partial ankylosis of both hips in a young man, *Clin.* 95; discussion (p. 95): Mr. Hastings, Mr. A. Pearce Gould, Dr. F. Parkes Weber
- , laryngeal paralysis following partial removal of the thyroid gland, *Laryng.* 150; discussion (p. 150): Mr. H. Tilley, Dr. F. Powell, Dr. Watson Williams, Dr. D. McKenzie, Mr. Horsford, Dr. D. Grant, Mr. Hastings (reply)
- , rupture of the tunica vaginalis in hydroceles, *Surg.* 165; discussion (p. 177): Mr. Godlee, Mr. Hastings (reply)
- HAWARD, J. WARRINGTON.—Discussion on treatment of inoperable sarcoma by mixed toxins, *Surg.* 44

- HAWKINS, F. H.—Discussion on congenital pulmonary stenosis, *Child*. 150
 —, malignant endocarditis of the tricuspid valve in a child aged 6, *Child*. 139; discussion (p. 142): Dr. Bellingham Smith, Dr. Cautley, Dr. Carr, Dr. Hawkins (reply)
 Hay fever, bacterial agency in connexion with, *R.S.M.* 15, 16
 Hay's pharyngoscope, *Otol.* 31
 —, demonstration of (H. J. Davis), *Laryng.* 23
 —, — (W. Milligan), *Otol.* 9
 HEAD, HENRY, F.R.S.—Cerebral hæmorrhage from luetic vessels, *Neur.* 31; discussion (p. 31): Dr. Head, Dr. Harry Campbell, Dr. Harris, Dr. James Taylor
 —, congenital lues causing optic atrophy and ultimately leading to dementia paralytica juvenilis, *Neur.* 33
 —, discussion on case of clicking noise in left ear, *Clin.* 94
 —, — of symmetrical trophic lesions in child (syringomyelia, Morvan's type), *Clin.* 103
 —, on cases of cerebral thrombosis, *Neur.* 87
 —, — on two cases of nervous disease of undetermined nature occurring in brother and sister, *Neur.* 96
 —, — on examination of blood-serum of idiots by Wassermann reaction, *Neur.* 123
 —, syphilis of the nervous system within six years of infection, *Neur.* 49; discussion (p. 60): Dr. Gordon Holmes, Dr. Wilfred Harris, Dr. Grainger-Stewart, Dr. Stanley Barnes
 —, thrombosis of cerebral arteries, *Neur.* 30; for discussion see *Head, Henry*, cerebral hæmorrhage from luetic vessels
 Head, injury to, extensive in boy (C. A. S. Ridout), *Child*. 162
 —, —, followed by arterio-venous aneurysm, with exophthalmos (F. S. Eve), *Clin.* 77
 —, relative proportion to pelvis, *Obst.* 114, 126, 127
 Headache accompanying tumours of pineal body, *Neur.* 71, 72
 Health, maintenance of, importance of reciprocal action of internal secretions as factor in, *Baln.* 11
 Heart, congenital malformation of, case (T. R. Whipham), *Child*. 149
 —, dilated, puncture of, *Med.* 81, 85
 —, —, in pericarditis, *Med.* 81
 —, dilatation of, diagnosis from pericardial effusion, *Med.* 62, 100, 101, 102
 —, — and hypertrophy of left side of, *Obst.* 215
 —, in pericarditis, *Med.* 56, 57, 69
 —, in rheumatic pericarditis, *Med.* 77
 —, of, without pericarditis, *Med.* 95
 —, of (general), results of pericarditis, *Med.* 56, 69
 —, severe, complicating case of prolapse of uterus (J. Inglis Parsons and T. C. Clare), *Obst.* 104
 —, —, treatment by ether inhalation without admixture of exhalations from lungs, *Clin.* 213, 215
 —, disease of, choice of anæsthetic for subjects of, *Anæsth.* 71, 72
 —, —, congenital œdema with cardiac defect in mother and daughter (F. J. Poynton), *Clin.* 66
 —, —, fats in treatment of, *Med.* 33, 34
 —, displacement of, with fibroid lung (Sir J. F. H. Broadbent, Bart.), *Clin.* 133
 —, —, in two cases of lobar pneumonia (W. P. Herringham), *Clin.* 25
 —, dorsal percussion of (Piorry), *Med.* 216
 —, fatty degeneration, *Child.* 82, 83
 —, left ventricle, dilatation in rheumatic pericarditis, *Med.* 69, 70
 —, of infant showing nodules on endocardium (R. C. Jewesbury), *Child.* 45
 —, malformation of, congenital, showing complete atresia of pulmonary artery (A. C. D. Firth and E. I. Spriggs), *Child.* 49
 —, sinking back of, in pericardial effusion, *Med.* 57, 58, 81
 —, valvular disease of, complicating fibromyoma of uterus, *Obst.* 215
 Heart-action and respiration, failure of, under anæsthetic associated with engorgement of thyroid gland, *Anæsth.* 45, 55

- Heart-block in acute rheumatism, Therap. 103
 — in severe influenza, Therap. 113
 Heart-dullness, increased, in pericarditis, preceded by compression of lower lobe of left lung.
 Med. 83, 84
 —, pear-shaped, Med. 78
 —, position of, in pericarditis, Med. 57, 58, 70
 Heart-massage, history of, Anæsth. 1
 — in heart-failure during anæsthesia (V. B. Orr), Anæsth. 1
 —, various methods, Anæsth. 6
 Heart-sounds, distant and muffled, physical sign of lymphatism, Anæsth. 52
 Heat as cause of summer diarrhoea, Epid. 139
 HEATH, P. MAYNARD.—Case of myositis ossificans traumatica, Clin. 149
 —, cystic swelling at the root of the nose, Child. 71
 Heberden's nodes in osteo-arthritis, Baln. 101, 103
 Hecht's reaction in diagnosis of syphilis, Surg. 220, 221, 227, 228, 239
 — compared with Wassermann's, Surg. 221, 239, 240
 — (modified) in diagnosis of syphilis, Surg. 220, 239, 240
 Hedley, J. P., fibroid of the cervix and body of uterus, Obst. 189
 Heiberg's modification of Gross's test for pancreatic insufficiency, Med. 201
 Held, development of neurone, Path. 4
 —, observations on neurone theory, quoted, Path. 28
 HELLIER, J. B.—Case of adenoma malignum cysticum cervicis uteri, Obst. 100; report of
 Pathology Committee (p. 103); discussion (p. 103): Dr. Cuthbert Lockyer
 Helmsley, epidemic diarrhoea at, Epid. 26
 Helouan as a health resort, Baln. 123
 Hemianæsthesia of left side, and anæsthesia of fifth nerve caused by pontine thrombosis
 (H. Campbell Thomson), Neur. 79
 Hemidystrophy, congenital (J. Howell Evans), Child. 23
 Hemihypertrophy, hypertrophy of type of, Child. 44
 Hemiparesis, transient, in cerebral syphilis, Neur. 52
 Hemiplegia frequent result of syphilis within six years of infection, Neur. 53
 —, left, and left third nerve paralysis (J. Taylor), Neur. 18
 — and paraplegia following syphilitic infection, Neur. 59
 Henoch's purpura, see *Purpura (Henoch's)*
 Hepatic dullness, Med. 226
 Hepato-adenoma, Path. 151
 Herbivora, tooth-follicle, important structure in, Odont. 162
 Heredity in cataract (familial discoid—or "Coppock"), Child. 68
 — in susceptibility to dental caries, Odont. 79
 — in telangiectases and epistaxis, Clin. 109, 110
 — in unidactyly, Child. 42
 HERMAN, G. E.—Discussion on case of myomectomy during pregnancy, Obst. 166
 —, discussion on Cæsarean section in eclampsia gravidarum, Obst. 203
 — on modern methods of delivery in contracted pelvis, Obst. 121
 —, statistics of puerperal eclampsia from Glasgow Maternity Hospital, Obst. 201
 HERN, W.—Discussion on absorption of teeth, Odont. 18, 19
 —, discussion on bacteriology of pyorrhœa alveolaris, Odont. 66
 — on case of painful attrition, Odont. 120
 — on condition of the mouth in chronic disease, Odont. 104
 — on exostoses of the mandible, Odont. 24, 27
 — on spindles of von Ebner, Odont. 149
 — on sterilizable film-holder, Odont. 89
 — on susceptibility of teeth to dental caries, Odont. 80, 82
 — on treatment of dental cysts invading the antrum, Odont. 118
 — on unerupted teeth and film radiographs, Odont. 89

- HERN, W.—Discussion on vertical fracture of mandible and canine tooth, *Odont.* 93
 —, relation of dentistry to other branches of medicine and its bearing upon the public health (presidential address, *Odontological Section*), *Odont.* 1-8
- Hernia, strangulated, reduction without laparotomy, mortality high after, *Child.* 173
- HERON, G. A.—Discussion on cases of epithelioma of larynx shown after operation, *Clin.* 118
 — discussion on hospital infection of tuberculosis, *Med.* 159
- HERRINGHAM, W. P.—Case of cyanosis with venous obstruction, *Clin.* 168; discussion (p. 169): Dr. Parkes Weber
 —, case of rheumatoid arthritis with albuminuria, and with great enlargement of the liver, *Clin.* 61; discussion (p. 62): Dr. W. P. Herringham, Dr. Poynton, Dr. Parkes Weber, Sir Dyce Duckworth, Dr. Rolleston, Dr. Herringham (reply)
 —, discussion on pericardial effusion, *Med.* 81
 — on scleroderma and sclerodactyly, *Clin.* 34
 — on thoracic aneurysm in a boy, *Clin.* 36
 — on treatment by radium of malignant growths, *Clin.* 166
 —, further notes on a case of neuritis of brachial plexus, *Clin.* 28
 —, sex incidence in gastric ulcer, *Surg.* 205
 —, two cases of lobar pneumonia with considerable displacement of the heart, *Clin.* 25; discussion (p. 27): Dr. Ewart, Dr. Herringham (reply)
- HERSCHELL, G.—Discussion on examination of intestines by X-rays, *Electr.* 120
 —, the therapeutical value of the lactic-acid bacillus, opening of discussion, *Therap.* 51; discussion continued by (p. 56): Dr. Goodbody, Dr. Vaughan Harley, Dr. Bulloch, Dr. Eyre, Dr. Robert Hutchison, Dr. Miller, Dr. Hertz, Dr. Gordon Lane, Dr. Langdon-Brown, Dr. Herschell (reply)
- HERTZ, A. F., bismuth oxychloride in examination by Röntgen rays, *Electr.* 110
 —, discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 100
 — on examination of intestines by X-rays, *Electr.* 120, 122
 — on pancreatic reaction and fecal analysis, *Med.* 204
 — on rôle of fats in treatment of disorders of stomach, *Med.* 35
 — on therapeutical value of lactic-acid bacillus, *Therap.* 62
 — on treatment of gastric ulcer, *Therap.* 134
 —, and ADAMS, F. S.—Formation of cholesterol gall-stones containing typhoid bacilli within sixty-eight days of the onset of typhoid fever, *Clin.* 169; discussion (p. 171): Mr. G. Wright
 —, see also *Clarke, S. A.*, case of phlegmonous gastritis
- HETT, G. S.—Attachment to von Brünings' instrument for facilitating manipulations under direct laryngoscopy, *Laryng.* 3
 —, case of extrinsic carcinoma of the larynx, *Laryng.* 53; discussion (p. 53): Dr. W. Hill, Dr. Scanes Spicer, Dr. FitzGerald Powell, Mr. Herbert Tilley, Dr. StClair Thomson, Mr. Trotter (reply)
 —, cases of neoplasm of the tonsil, *Laryng.* 2
 —, chronic glanders in a man aged 24, *Laryng.* 1; discussion (p. 1): Dr. W. Hill, Dr. F. Powell, Mr. Tilley, Mr. Hett (reply)
 —, discussion on case of tuberculosis of pharynx, *Laryng.* 59
- HEWITT, F. W., M.V.O.—Discussion on lymphatism, *Anæsth.* 40
- Hewitt, Gordon, on flies, alluded to, *Epid.* 154
 —, transmission of typhoid fever by flies, quoted, *Epid.* 213, 214
- HEWLETT, Professor R. T.—Discussion on vaccine therapy, *R.S.M.* 84
 —, effect of injection of various tuberculins and of tubercle endotoxin on opsonizing action of serum of healthy rabbits, *Path.* 165
- Hexamethylene-tetramin and pure ether, inhalations of, in treatment of laryngeal tuberculosis, *Laryng.* 39
- Hiatus semilunaris, removal of hypertrophied anterior lip of, for long-standing catarrh (J. Dundas Grant), *Laryng.* 131
- HIGGS, F. W.—Discussion on case of congenital hemidystrophy, *Child.* 24

- Higgs, F. W.—Discussion on case for diagnosis, Child. 24
 —, discussion on specimen of urinary organs with presence of diphtheritic membrane in trachea, Child. 91
 —, specimens from a case of lymphosarcomatosis with clinical and post-mortem notes, Child. 17
 High-frequency currents, rectal administration in relief of symptoms of prostatic obstruction, Electr. 142
 —, cases described, Electr. 143-145
 —, technique in gynaecological applications, Electr. 35
 — spark in treatment of nævi, Electr. 40
 High-tension transformer, with valve tubes, value of, in production of X-rays, questioned, Electr. 150, 151
 —, rectifying, value of, in production of X-rays, Electr. 152, 153
 —, compared with that of induction coil, Electr. 152, 153
 — (Snook's type), effect upon tubes, Electr. 158
 —, noise a drawback, Electr. 156, 159
 —, screen examinations under, Electr. 158, 159
 —, value of, in production of X-rays, Electr. 155-158
 Hill, Professor A., "The Body at Work," quoted, Odont. 156
 HILL, WILLIAM.—Benign laryngeal growth, Laryng. 94; discussion (p. 94): Dr. Pegler, Dr. Dundas Grant
 —, case of carcinoma of gullet in a man aged 62, Laryng. 120
 —, cases of malignant disease treated by radium, Laryng. 39
 — case of traumatic cicatricial stenosis of larynx in a child aged 6, Laryng. 120
 —, discussion on case of extrinsic carcinoma of larynx, Laryng. 53
 — of laryngeal new growth for diagnosis, Laryng. 61
 — of pemphigus vegetans affecting larynx and oral cavity, Laryng. 78
 — of rare congenital deformity of nose, Laryng. 85
 — on chronic glanders in a man, Laryng. 1
 — on endothelioma of ethmoid, Laryng. 67
 — on foreign bodies in lung and œsophagus, Laryng. 76
 — on treatment by radium of malignant growths, Clin. 166
 — of tuberculous laryngitis by galvano-puncture, Laryng. 122
 — on tuberculosis of larynx in girl, Laryng. 128
 — on vulsellum-catch forceps, Laryng. 125
 —, endolaryngeal carcinoma, Laryng. 168
 —, epithelioma of nasal vestibules and adjacent areas: recurrence after surgical removal: radium treatment commenced, Laryng. 26
 —, hypertrophic laryngitis with stenosis in a syphilitic subject, Laryng. 93; discussion (p. 98): Dr. Dundas Grant, Mr. Wilkinson, Dr. Hill (reply)
 —, improved direct-vision laryngoscope for endolaryngeal operations, Laryng. 26
 —, large chronic swelling in right tonsillar and epitonsillar region, with enlarged gland in the neck, ? sarcoma, Laryng. 132; discussion (p. 133): Dr. J. Dundas Grant, Mr. H. Tilley, Dr. Donelan, Dr. W. Hill (reply)
 — globular dilation of the upper third of the œsophagus above a malignant structure, Laryng. 165; discussion (p. 165): Dr. S. Spicer
 —, party-wall pharyngeal cancer, Laryng. 142; discussion (p. 142): Mr. A. Evans, Mr. Westmacott, Dr. H. J. Davis, Dr. Dundas Grant
 — skiagrams of cases of carcinoma of gullet, Laryng. 120
 —, two cases of malignant disease of neck undergoing treatment by radium, Laryng. 25
 — and FINZI, N. S.—Case of œsophageal growth under treatment by radium, Clin. 160; discussion (p. 161)
 —, malignant growth in neck treated with radium, with recurrence in mediastinum, Clin. 159
 Hinton, James, "Mystery of Pain," quoted, Obst. 2

- Hip-joint, tuberculosis of, treatment with tuberculin by aid of opsonic index, R.S.M. 47
 ———— with vaccine, R.S.M. 105
- Hip-joints, partial ankylosis of both, with bending of bones of both legs (S. Hastings), Clin. 95
- Hippocrates, on experience and judgment, quoted, R.S.M. 113
- Hirschsprung's disease, see *Colon (Dilatation, idiopathic)*
- His, origin of nerve-cell, Path. 3
- Hoarseness, case of, in woman (G. C. Cathcart), Laryng. 56
 ————, treatment by inhalation of compound tincture of benzoin, Laryng. 56
 ————, two cases of (E. W. Roughton), Laryng. 40
- Hodara, pathology of acne, Path. 172
- Hodgkin's disease, see *Lymphadenoma*
- HODGSON, R. H.—Case of pulmonary tuberculosis with large cavity treated by a new method; also notes of other cases in which ether was tried, Clin. 213
 ————, discussion on case of laryngeal growth, Laryng. 39
 ———— on placenta and membranes of quadruplets, Obst. 80
 ————, notes on an apparatus for the administration of ether by the purely open method, Anaesth. 16; discussion (p. 18): Mr. R. Gill
- Hoffa, osteo-arthritis, quoted, Baln. 90, 91
- HOLLAND, C. T.—Discussion an examination of intestines by X-rays, Electr. 120
 ————, discussion on radium in treatment of malignant growths, Electr. 69
 ———— on X-ray treatment of lymphadenoma, Electr. 132
 ————, points in the diagnosis of ureteral calculi, Electr. 87-97
- Holmes, E. M., botanical identification of kangalugi root, quoted, Therap. 67
- HOLMES, G.—Case of (1) cerebellar sclerosis, (2) olivo-ponto-cerebellar atrophy, Neur. 89
 discussion (p. 90): Dr. F. Buzzard, Dr. F. E. Batten, Dr. S. A. K. Wilson, Dr. G. Holmes (reply)
 ————, discussion on cases of cerebral thrombosis, Neur. 86
 ———— chronic syphilitic poliomyelitis and chronic meningomyelitis, Neur. 26
 ———— on distal type of myopathy, Neur. 94
 ———— on syphilis and parasymphylis of the nervous system, Neur. 60
 ———— on tumours of pineal body, Neur. 78
 ———— and May, W. Page, exact origin of pyramidal tracts, quoted, Path. 72
- Holmes, O. Wendell, "Autocrat of the Breakfast-table," quoted, R.S.M. 157
- Homalomyia*, breeding-places of, Epid. 143
- Home management, training in, Epid. 39
- HOPEWELL-SMITH, A.—Case of infective disease of the jaws associated with the absorption of the teeth, Odont. 9-17; discussion (p. 18): Mr. Hern, Mr. F. J. Bennett, Mr. H. Baldwin, Mr. Clement Lucas, Mr. S. Mummery, Mr. Hopewell-Smith (reply)
 ————, two odontoceles and some other cysts, Odont. 121-142
- HORDER, T. J.—Diagnosis of endocarditis, quoted, Therap. 101
 ————, discussion on vaccine therapy, R.S.M. 139
- HORNE, W. JOBSON.—Discussion on bands between Eustachian tube and pharynx, Laryng. 69
 ————, discussion on case of chronic laryngitis with (?) prolapse of ventricle on right side, Laryng. 31
 ———— of healed tuberculous laryngitis treated by galvano-puncture, Laryng. 121
 ———— of malformation of soft palate and uvula, Laryng. 131
 ———— of tuberculosis of pharynx, Laryng. 59
 ———— on endothelioma of ethmoid, Laryng. 67
 ———— on model demonstrating "back" and "belly" breathing, Laryng. 50
 ———— on papillomata of larynx from boy removed by direct method, Laryng. 97, 98
 ———— on papillomata of larynx with symmetrical papillomata on the palate, Laryng. 95
 ———— on specimen of larynx showing re-development of epithelioma on left cord after removal of right for same cause, September, 1896, Laryng. 35
 ———— on tuberculosis of larynx in girl, Laryng. 127

- HORNE, W. JOBSON.—Discussion on vulsellum-catch forceps, *Laryng.* 124
 —, series of specimens and a case illustrating diseases of the ventricle of the larynx, *Laryng.* 157; discussion (p. 158): Dr. S. Spicer, Dr. J. Dundas Grant, Dr. Horne (reply)
- HORSE, tick-bites on, eosinophile cells, on exudate from (J. B. Cleland), *Path.* 42
- HORSES, effect of ingestion of, or abstinence from, common salt upon, *Baln.* 61, 62
- HONSFORD, C.—Discussion on case of chronic laryngitis with (?) prolapse of ventricle on right side, *Laryng.* 31
 —, discussion on case of "Singer's node" on left cord, *Laryng.* 108
 — on hypertrophy of lingual tonsil, *Laryng.* 156
 — on laryngeal paralysis, *Laryng.* 151
 — on model demonstrating "back" and "belly" breathing, *Laryng.* 48
 — on treatment of tuberculous laryngitis by galvano-puncture, *Laryng.* 122
 — on tuberculosis of larynx in girl, *Laryng.* 128
- HORSLEY, Sir V., F.R.S.—Cerebral operations for organic disease causing special sense discharges, quoted, *Neur.* 3, 10, 13, 16
 —, discussion on cases of cerebral thrombosis, *Neur.* 86
 — on tumours of pineal body, *Neur.* 77
 — and Clarke, R. H., on fibres entering cerebellum, quoted, *Path.* 65
- HORT, E. C., toxic products of tissue-degeneration, *R.S.M.* 85
- HOSPITAL infection of tuberculosis as exemplified by the records of Mount Vernon Hospital (J. E. Squire), *Med.* 137-156
- HOSPITALS, communicability of typhoid fever to non-fever patients in, discussed, *Epid.* 108
 —, incidence of diarrhoea on those in contact with sick in, *Epid.* 105
 —, infection of patients in, with diarrhoea, *Epid.* 106, 107, 108
 —, prevention of overcrowding in out-patient departments, *Epid.* 34
 —, teaching of therapeutics in the hospital wards (Sir T. C. Allbutt), *Therap.* 1-16
 — (public) in Manchester, causes of death occurring in, *Epid.* 18, 19
 — in Manchester, percentages of deaths occurring in, *Epid.* 18
- HOT-air bath, in chronic nephritis with threatening uræmia, *Therap.* 147
- "Hour-glass" stomach, *Electr.* 113
- HOUSE-FLIES, capture by beer-traps, *Epid.* 169
 — methods of, *Epid.* 141, 142, 260
 — by stick papers, *Epid.* 169
 —, conveyance of micro-organisms by, *Epid.* 261
 —, destruction by *Empusa musca*, *Epid.* 154, 166
 —, effect of rainfall and temperature on, *Epid.* 161
 —, as factors in transmission of summer diarrhoea, *Epid.* 138-141
 —, flight of, *Epid.* 260
 —, life history of, *Epid.* 143, 154-157
 —, numbers captured in Manchester compared with deaths from diarrhoea, *Epid.* 148-149
 —, prevalence in Manchester, correction of curves, *Epid.* 170-174
 —, fall of curve in, *Epid.* 166
 —, relation to fatal cases and to prevalence of summer diarrhoea, *Epid.* 141, 145-49, 169, 270
 — to onset of cases of typhoid fever, *Epid.* 145-149
 — to prevalence of typhoid fever, *Epid.* 175, 178, 179
 — and privy-middens in connexion with prevalence of typhoid fever, *Epid.* 187
 —, transmission of summer diarrhoea by, *Epid.* 157-159, 266-67
 — of typhoid fever by, *Epid.* 138, 212-215, 262, 263, 265, 266, 267, 268
- HOUSE-TO-HOUSE inquiry to estimate degree of poverty, *Epid.* 3, 12
- HOVELL, T. MARK.—Discussion of papillomata of larynx from boy, removed by direct method, *Laryng.* 98
 —, discussion on treatment of case of tuberculosis of larynx, *Laryng.* 153

- HOWARD, HEATON C.—Stricture of the rectum treated by fibrolysin, Clin. 87; discussion (p. 88): Mr. Pearce Gould, Dr. Leonard Williams, Professor William Osler, F.R.S., Mr. Carless, Mr. Herbert Tilley, Mr. Arthur Barker, Dr. Essex Wynter
- HOWARTH, W.—Discussion on treatment of case of tuberculosis of the larynx, Laryng. 153
- , tuberculosis of the larynx with considerable dysphagia treated and relieved by congestion hyperemia, Laryng. 164; discussion (p. 164): Dr. J. Dundas Grant, Mr. Howarth (reply)
- HOWELL, C. M. HINDS.—Discussion on case of compression paraplegia: syphilitic meningitis, Neur. 22
- , discussion on cases of cerebral thrombosis, Neur. 85
- , secondary growths affecting spinal roots (by E. Farquhar Buzzard), pathological report, Neur. 125
- , tumours of pineal body, Neur. 65; discussion (p. 77): Sir Victor Horsley, Dr. G. M. Holmes, Dr. Howell (reply)
- Huggard, W. R., on Naples as winter station, quoted, Baln. 28
- HUGGINS, MARTIN, and CORNER, EDBED M.—Pyæmic knee- and shoulder-joints treated by tapping and incision, Clin. 120; discussion (p. 121): Mr. A. E. Barker, Dr. Parkes Weber
- Hughlings Jackson lecture, special discharges from organic disease (Sir W. R. Gowers), Neur. 1
- HUGO, H. F. L.—Statistics of pericardial effusion from Victoria Hospital for Children, Chelsea, Med. 119
- Hulke, J. W., leukoplakia, quoted, Obst. 29
- Hulst's electro-static machines, Electr. 80
- Humerus, cyst of, diagnosis from sarcoma, Child. 99
- , fracture, operation (H. Lett), Child. 98
- , dislocation, recurring, detachment of tendon of subscapularis muscle for (W. G. Spencer), Clin. 20
- , fracture of, at site of innocent cyst (R. C. Elmslie), Clin. 192
- (right) and left femur, shortening of, due to unobserved fractures in early life (H. Burrows), Child. 165
- Humidity, controls subjective effects and immediate results of climate, Baln. 3, 4
- HUMPHRIS, F. HOWARD.—Discussion on arthritis deformans, Baln. 97
- , discussion on electrical treatment of prostatic eruption, Electr. 147
- , on points for the use of and indications for electrotherapy in some gynaecological affections, Obst. 26
- , on therapeutical use of statical electricity, Electr. 84, 85
- , the static wave current, Electr. 97-104; discussion (p. 104): Dr. Sloan, Dr. Horace Manders, Dr. E. Wagstaff, Dr. Curtis Webb, Dr. Humphris (reply)
- HUMPHRY, R. E.—Discussion on lymphatism, Anæsth. 51
- Hunger-pain in duodenal ulcer, Surg. 70, 94, 97, 98, 99, 100, 102, 112
- Hunt, Dr., experiments with streptococcus agglutination, R.S.M. 118
- HUNT, J. MIDDLEMASS.—Discussion on condition of the mouth in chronic disease, Odont. 105
- , discussion on specimen of larynx showing re-development of epithelioma on left cord after removal of right for same cause, September, 1896, Laryng. 34
- Hunter, John, rupture of tunica vaginalis in hydroceles, quoted, Surg. 165
- HUNTER, WALTER K.—Acute degenerative changes in the nervous system as illustrated by snake-venom poisoning, Neur. 105-115; discussion (p. 116): Dr. J. A. Ormerod, Dr. Wilfred Harris, Dr. Hunter (reply)
- Hutchinson, sclerodactylia and acroteric scleroderma of, associated with previous family history of angioneurotic œdema (T. D. Savill), Clin. 33
- HUTCHINSON, JONATHAN.—Discussion on present position and treatment of syphilis, Surg. 242
- , discussion on radical operation for cancer of pylorus, Surg. 161
- HUTCHISON, R.—Achondroplasia in a twin, Child. 41; discussion (p. 42): Dr. Shuttleworth, Dr. Chapman, Dr. Garrod, Dr. Hutchison (reply)
- , case of speech defect, Child. 129; discussion (p. 129): Dr. Hutchison, Dr. Shuttleworth, Dr. Langmead, Dr. Hutchison (reply)

- HUTCHISON, R.—Case of *unidactyly*, Child. 41; discussion (p. 42): Dr. Shuttleworth, Dr. Chapman, Dr. Sutherland, Dr. Hutchison (reply)
- , *cirrhosis of liver*, Child. 40; discussion (p. 42): Dr. Parkinson, Dr. G. A. Sutherland, Dr. Hutchison (reply)
- , discussion on case of early *lymphadenoma*, Child. 135
- on diagnosis and treatment of *duodenal ulcer*, Surg. 97
- on *oxaluria* and *calcium-oxalate deposit*, Med. 21
- on rôle of *fats* in treatment of disorders of stomach, Med. 36
- on teaching of therapeutics in hospital wards, Therap. 11
- on therapeutic value of *lactic-acid bacillus*, Therap. 60
- , on consumption of salt, quoted, Baln. 64
- , three cases of *oxycephaly*, Child. 125; discussion (p. 127): Dr. Morley Fletcher, Dr. Sutherland, Dr. F. Parkes Weber, Dr. G. E. Shuttleworth, Dr. Hutchison (reply)
- Hydatid cyst of liver*, painless, Obst. 5
- Hydrobilirubin*, reaction for, Med. 198, 199
- Hydrocele*, rupture of *tunica vaginalis* in (S. Hastings), Surg. 165
- Hydrocephalus* (internal) and *amaurosis* without definite ophthalmoscopic changes, following symptoms of posterior basic meningitis or *ependymitis* (F. Parkes Weber), Child. 62
- Hydrochloric acid*, diminution of secretion in prevention of *duodenal ulcer*, Surg. 105
- , poisoning by, causing *phlegmonous gastritis* (S. A. Clarke, A. F. Hertz, and R. P. Rowlands), Clin. 209
- Hydrotherapeutics*, origin of, empiric, Baln. 6
- Hydrothorax*, subjects of, in childhood, insurability of lives, Surg. 55
- Hygiene*, promotion of, by prevention of idleness among working men, Epid. 42
- Hygroma*, cystic, extensive operation for (C. P. Childe), Child. 157
- , multilocular cystic, of neck (C. P. Childe), Child. 157
- Hyoid bone*, fracture of ("garrotter's throat"), (H. J. Davis), Laryng. 77
- Hyperacidity* in gastric ulcer, Med. 119, 124, 126, 132, 135, 137
- Hyperemia* (active), induction of, to test progress of extinction of bacterial infection, R.S.M. 23
- (congestion): *tuberculosis of larynx*, with considerable *dysphagia*, treated and relieved by (W. Howarth), Laryng. 164
- Hyperesthesia sexualis*, Neur. 48
- Hyperchlorhydria*, Surg. 72
- , essentially *duodenal ulcer*, Surg. 74
- , geographical distribution in Germany, Surg. 101
- , hypersecretion, Med. 26, 31
- in case of *duodenal ulcer*, Surg. 195, 203
- in disease of appendix, Surg. 201
- , possible cause of *pancreatitis*, Med. 170
- relation to *duodenal ulcer*, Surg. 99, 101, 108
- Hyperpyrexia* in *pneumococcal colitis*: hemorrhage treated by appendicostomy (H. E. Bruce-Porter, J. P. Lockhart Mummery, and W. Hale White), Clin. 48
- Hypersecretion* and connexion with latent disease of appendix (W. Soltan Fenwick), Surg. 177
- , biliary, frequency of, Surg. 184
- , degrees of, Med. 26, 31
- (gastric), effect of appendicectomy on, Surg. 190
- , and symptoms of appendicular disease, Surg. 206
- Hypertrophy of hemihypertrophy type*, Child. 44
- Hypochondriasis* and incipient disease of appendix, Surg. 184
- Hysterectomy*, abdominal, for pregnancy in a septate uterus (W. Tate), Obst. 190
- , —, —, sixty-three hours after labour, for necrosed and suppurating subperitoneal fibroid (P. D. Turner), Obst. 61
- (total), following posterior *Cæsarean section*, for fibroids (H. R. Spencer), Obst. 82
- Hysteria*, and incipient disease of appendix, Surg. 184

- Hysteria, misleading term in gynæcological diagnosis, Obst. 3
 —, subjects of, dermatitis artefacta in, Derm. 28, 29
 —, —, —, always accompanied by anaesthesia of soft palate, Derm. 29
 Hysteropexis hypogastrica, see *Ventro-fixation*
 Hysterotomy, see *Cæsarean section, extra-peritoneal*
- Ice-bag, application in treatment of rheumatic pericarditis, Med. 71
 Ichthyosis, generalized, keratosis associated with (H. G. Adamson), Derm. 83
 — and scarlet fever, Epid. 63
 Icterus, see *Jaundice*
 Idiocy, amaurotic family, case of (E. Bellingham Smith), Child. 148
 — — —, without characteristic ophthalmoscopic signs (F. Parkes Weber), Child. 58
 — — — and internal hydrocephalus, without definite ophthalmoscopic changes, following symptoms of posterior basic meningitis or ependymitis (F. Parkes Weber), Child. 62
 —, congenital syphilis in subjects of, Neur. 119, 120, 122
 — and syphilis, causal relation between discussed, Neur. 120, 122
 Idiots, blood-serum of, examination by Wassermann reaction (H. R. Dean), Neur. 117
 —, examination of cerebrospinal fluid of, by Wassermann reaction, Neur. 118, 123
 Idleness, cause of pauperism, Epid. 8
 —, prevention of, among working men, hygienic importance, Epid. 42
 Ignorance, cause of pauperism, Epid. 9
 Ileo-colic prolapse, remaining after spontaneous reduction of intussusception, Child. 176
 Ileo-colitis, diagnosis from intussusception, Child. 168
 ILES, A. J. H.—Simple X-ray steroscope, Electr. 138; discussion (p. 139): Dr. R. Morton
 Iliac region, new field for diagnosis opened up by dorsal percussion, Med. 238
 — — — — —, important bearing on diagnosis of appendicitis, Med. 238
 — — — — —, right, origin of pain in, Obst. 6, 7
 Ilium, sarcoma of, treatment by Coley's fluid (A. Carless), Clin. 113
 Illness, cause of pauperism, Epid. 8
 Immunity, estimation of cell as guide to, R.S.M. 74
 — — —, serum-examination as guide to, R.S.M. 74
 — — —, increase of, due to lessening of intoxication, R.S.M. 122, 123
 — — —, production of, in vaccine therapy, R.S.M. 196
 — — — (active), feature of vaccine therapy, R.S.M. 212, 213
 — — — (passive), limitations of, R.S.M. 213
 Immunity-reactions to bacteria, Therap. 78
 — — — — — to cancer (E. F. Bashford), Therap. 69-81
 Imperial Cancer Research Fund, laboratory of, varieties and sub-varieties of cancer under observation at, Therap. 74
 Incapacity, cause of pauperism, Epid. 8
 Incisors, mutilation of, in Egyptian skulls exhibited, question whether before or after death discussed, Odont. 154, 155
 India, ablation of ovaries in young practised in, Path. 118
 —, British Army in, prophylaxis of syphilis, Surg. 226
 —, typhoid fever in, transmission by flies, Epid. 262, 263
 India-rubber gloves, wearing of, at operations for delivery, Obst. 121, 122
 Indians, North American, abstinence from use of salt by, Baln. 61
 Indicanuria in acute pancreatitis, Med. 166
 — in chronic pancreatitis, Med. 167, 168, 170, 171, 172, 173, 174
 —, treatment by application of lactic-acid bacillus, Therap. 62
 Indigo, passage through small intestine, Med. 204
 Induction coil, value of, in production of X-rays, Electr. 151, 152
 — — — — —, compared with that high-tension rectifying transformer, Electr. 152, 153

- Industrial organization, waste of, *Epid.* 37, 41
- Infancy, acute empyema of, prognosis in, *Surg.* 53
- , malnutrition in, connexion of hypoplasia of enamel with, *Odont.* 79, 82, 84
- , myelogenous leukemia in, case of (T. R. Whipham), *Child.* 92
- , —, —, rarity of, *Child.* 93, 94
- , infant, heart of, showing nodules on endocardium (R. C. Jewesbury), *Child.* 45
- , —, microscopic section from liver of case of icterus in (G. Carpenter), *Child.* 48
- , —, pneumococcal infection in simulating generalized tuberculosis (J. Walter Carr), *Child.* 14
- , —, rare congenital deformity of nose in (G. Wilkinson), *Laryng.* 83
- , —, strangulation of small intestine by band in (J. P. Lockhart Mummery), *Child.* 37
- Infantilism (pancreatic), pancreatic reaction in subjects of, *Med.* 177
- Infants, bottle feeding of, *Odont.* 7
- , —, breast feeding of, *Odont.* 6
- , —, diarrhoea among, at Manchester, deductions from fatal cases, *Epid.* 103
- , —, distribution of summer diarrhoea among, *Epid.* 136
- , —, dying from various disorders, question and significance of muco-pus in middle ears of, *Otol.* 26, 27
- , —, instruction to mothers amongst poorest classes as to care of, *Epid.* 34
- , —, malignant diphtheria in, case of (J. D. Rolleston), *Child.* 74
- , —, —, epidemics recorded, *Child.* 75
- , —, —, newly-born, death-rate of, *Obst.* 232
- , —, —, pneumococcal pericarditis in, suggested origin, *Med.* 89
- , —, —, scarlet fever in, isolation unnecessary, *Epid.* 64
- , —, —, suffering from pneumonia, attacked with diarrhoea, *Epid.* 107
- , —, —, symptoms masking intussusception in, *Child.* 167
- Infection, liability to, during school life, *Epid.* 45
- , —, mode of, of various diseases, in schools, *Epid.* 62
- , —, products of, *R.S.M.* 161
- Infectious diseases in schools, code of rules for prevention of, *Epid.* 45, 46
- Infective diseases, discoveries in, the work of bacteriologists, *R.S.M.* 77, 78
- Inflammation, acute, associated with primary carcinoma of Fallopian tube (W. W. H. Tate), *Obst.* 75
- , —, indication for treatment by static wave current, *Electr.* 99
- , —, pain due to, felt when at rest, *Obst.* 6
- Inflation in reduction of intussusception in children, *Child.* 171, 175, 177
- Influence machine, electro-static, electrodes of, *Electr.* 75
- , —, —, —, —, (Suchier's), *Electr.* 73, 74
- , —, —, —, —, electrodes of, effects of separation, *Electr.* 75
- Influenza, epidemic, case complicated by acute encephalitis, *Neur.* 132
- , —, fats in treatment of, *Med.* 33, 34
- , —, general paralytics easily succumb to, *Neur.* 49
- , —, severe, heart-block in, *Therap.* 113
- , —, treatment by influenza vaccine, *R.S.M.* 69, 70, 73, 185
- , —, —, by pneumococcal vaccine, *R.S.M.* 70
- Ingestion, tuberculous infection by, *Child.* 27
- , —, —, —, —, compared with that by inhalation, *Child.* 27, 28
- Inglis, on absorption of roots of permanent teeth, quoted, *Odont.* 15
- Inguinal region, right, origin of pain in, *Obst.* 6, 7
- Inhalation, tuberculous infection by, compared with that by ingestion, *Child.* 27, 28
- Injuries, causation of malignant tumours by, how explained, *Surg.* 10, 11
- INMAN, A. C.—Discussion on vaccine therapy, *R.S.M.* 208
- , —, oral administration of tuberculin, quoted, *R.S.M.* 118
- , —, and Latham, A., oral administration of horse serum, quoted, *R.S.M.* 199
- Insanity, following development of clicking ties, *Clin.* 94, 95
- Insurability of lives the subjects of hydrothorax or empyema in childhood, *Surg.* 55

- Insurance against unemployment, *Epid.* 37
- Intellect^o, activity of, temporary, in general paralysis, *Neur.* 48
- Intemperance, cause of, among working men, *Epid.* 41
- cause of pauperism, *Epid.* 9
- Interarytenoid space and left vocal cord, tuberculosis of, healed by two months' silence and sanatorium treatment (StC. Thomson), *Laryng.* 6
- (left), epiglottis, and ary-epiglottic fold, tuberculosis of, healed by galvano-cautery and sanatorium treatment (StC. Thomson), *Laryng.* 7
- Interdepartmental Committee on Physical Deterioration, findings as to ill-results of dental disease, *Odont.* 5
- International Congress on Obstetrics and Gynecology, Geneva (1896), pronouncement as to Caesarean section, *Obst.* 198
- Intestine, prolapse of, through ectopia vesicæ (A. C. D. Firth), *Child.* 55
- , resection of, in intussusception, *Child.* 169, 170
- (small), intussusception of, containing sarcoma of intestinal wall; treatment by enterectomy; recovery (C. A. S. Ridout), *Child.* 161
- , passage of indigo through, *Med.* 204
- , resection of 31 in. for irreducible intussusception in young adult; recovery, *Child.* 173
- , strangulation by band in infant (J. P. Lockhart Mummery), *Child.* 37
- Intestines, carbohydrates in abnormal fermentation of, *Therap.* 55, 56, 57
- , carbohydrates in, abnormal fermentation tests for distinguishing from intestinal putrefaction of proteids, *Therap.* 55, 56, 57
- , cancer of, Röntgen-ray diagnosis, *Electr.* 116
- , diseases of, chronic pancreatitis secondary to, *Med.* 169
- , examination of, by Röntgen rays (F. M. Groedel), *Electr.* 109
- , invasion by pneumococcus and streptococcus, effect of lactic bacillus on, *Therap.* 60, 63
- , proteids in, abnormal putrefaction of, *Therap.* 55, 56, 57
- , —, cases dependent upon, benefited by lactic-bacillus administration, *Therap.* 54, 55
- , —, tests for distinguishing from intestinal fermentation of carbohydrates, *Therap.* 55, 56, 57
- , see also *Bacteria, intestinal*
- Intoxication, lessening of, due to increase of immunity, *R.S.M.* 122, 123
- Intranasal disease (obstruction, pressure, and ethmoidal suppuration) associated with aggravated, lifelong stammering (R. H. Scanes Spicer), *Laryng.* 119
- Intrapericardial pressure in pericardial effusion, *Med.* 60
- Intraspinal injections of magnesium sulphate solution in treatment of tetanus, *Med.* 39
- Intubation (per-oral), demonstration of Kuhn's instruments for (W. Milligan), *Otol.* 49
- Intussusception, apex of, formed by involution of acutely inflamed appendix into lumen of cæcum; operation; recovery (I. Back), *Child.* 25
- in children, age and sex incidence, *Child.* 166, 167
- , diagnosis (C. P. Childe), *Child.* 165-173, 174, 175
- , aids to, *Child.* 166, 167, 168
- from bleeding due to hæmorrhoids, *Child.* 177
- from ileo-colitis, *Child.* 168
- from Henoch's purpura, *Child.* 168, 173, 176
- , ileo-cæcal, *Child.* 166
- , irreducible, application of spinal anaesthesia in, *Child.* 175
- , recovery hopeless, *Child.* 169
- , prognosis, *Child.* 169, 170, 171, 176
- , reducible in early stages, *Child.* 169, 170
- , reduction by inflation, *Child.* 171, 175, 177
- , removal of (length, 14 in.), containing sarcomatous growth; recovery, *Child.* 175
- , statistics of mortality, and recovery after operation, *Child.* 170, 171

- Intussusception in children, symptoms, Child. 167
 ———, masking, Child. 167
 ———, tenesmus and passage of blood and mucus in, Child. 168
 ———, treatment (C. P. Childe), Child. 165-173
 ———, operative, by laparotomy and reduction of tumour, Child. 169, 170
 ———, by reduction without laparotomy, Child. 171, 177
 ———, tumour in, presence of discoverable by palpation, Child. 167, 168, 169, 170, 174
 ———, presence of, masked in later stages by general peritonitis, Child. 174
 ———, irreducible, in young adult, case and specimen exhibited after operation, Child. 173
 ——— of small intestine, containing sarcoma of intestinal wall; treatment by enterectomy; recovery (C. A. S. Ridout), Child. 161
- Inunction in scarlet fever, Epid. 81, 85
- Iodides in treatment of syphilis, Surg. 216, 225, 237, 245
- Iodipin in treatment of syphilis, Surg. 216, 236
- Ionic medication, technique in gynaecological applications, Electr. 33; Obst. 16, 17, 22, 23, 25, 26
 ———, with continuous current, Electr. 105
- Ionization in atmospheric electricity, Baln. 53
- Ions, passage of, in static electricity, Electr. 75
- Ipswich, diarrhoea in, diminution in prevalence, Epid. 112, 113, 117, 119
 ———, typhoid fever in, diminution in prevalence, Epid. 111, 113, 116
 ———, water-closet system prevalent in, Epid. 111, 115
- IREDELL, Dr.—Discussion on rodent ulcer treated by radium, Clin. 163
- Ireland, gout rare in, Clin. 24
 ———, seasonal prevalence of scarlet fever in early nineteenth century, quoted, Epid. 75, 88, 93
- Iris, tubercle of, treatment by tuberculin T.R., process following, R.S.M. 193
- Iritis (gonococcal), treatment by vaccine, R.S.M. 189
- Isolation in scarlet fever unnecessary, Epid. 58
- Isolation hospitals, classification of cases, Epid. 82
 ——— and arrangement of cases by barrier system, Epid. 83
 ——— by bed isolation system, Epid. 83, 84
 ——— by cubicle system, Epid. 82, 83, 89
 ———, control of scarlet fever in, Epid. 74, 80
 ———, under strict aseptic treatment, Epid. 80
 ———, cross-infection in, Epid. 79, 87, 94
 ———, disinfectant methods in, Epid. 84, 85
 ———, sterilizers in, Epid. 84
 ———, toilet of patients, Epid. 84
 ———, ward-space for children same as for adults, Epid. 84
- Isolysin reaction in cancer immunization, difficulty of obtaining, Therap. 77
- Jackson, J. Hughlings, olfactory discharges from organic disease, quoted, Neur. 2, 4
 ———, organic disease, cause of auditory discharges, Neur. 6
- JACOB, F. H.—Case of acholuric jaundice, Clin. 157
- JACOBS, Professor, statistics of ovarian growths quoted, Obst. 98
- JAMES, W. WARWICK.—Discussion on exostoses of the mandible, Odont. 26
 ———, discussion on odontocles and some other cysts, Odont. 159
- Jardine, R., on puerperal eclampsia, quoted, Obst. 201
- Jaundice, acholuric, case (F. H. Jacob), Clin. 157
 ——— (congenital), case of (L. G. Parsons), Child. 136
 ——— and biliary hypersecretion, Surg. 184
 ——— (catarrhal), Med. 206
 ——— and chronic pancreatitis, Med. 171
 ———, lymphoid swelling and, Med. 171

- Jaundice in infant, microscopical specimen of liver from case of (G. Carpenter), *Child.* 48
 — in phosphorus poisoning, *Therap.* 66
 — with areas of unpigmented skin in case of cancer of cardiac orifice of stomach (H. D. Rolleston), *Clin.* 195
- Javal and Widal on excretion of salt from and retention in body, *Baln.* 67, 68
- Jaw, cysts of, *Odont.* 158
 —, lower, unilateral overgrowth of (S. Boyd), *Odont.* 47
- Jaws, cysts of (A. Hopewell-Smith), *Odont.* 121-142
 — infective disease of, associated with absorption of teeth (A. Hopewell-Smith), *Odont.* 9-19
 —, tumours of, malignant, diagnosis by X-rays, *Odont.* 158, 161
 —, upper and lower, model of, and impression of roof of mouth from young man (A. R. Tweedie), *Laryng.* 141
- JELLETT, H.—The place of Caesarean section in the treatment of placenta prævia, *Obst.* 167-177; discussion (p. 177): Dr. H. Spencer, Sir F. H. Champneys, Dr. Purslow, Dr. A. Routh, Dr. W. J. Gow, Dr. W. S. Griffith, Dr. H. Macnaughton-Jones, Dr. H. Jellett (reply)
 —, sarcoma of the body of the uterus from a girl aged 20, *Obst.* 186
- JENKINS, G. J.—Some features of the auditory apparatus of a 16 mm. human embryo, as shown in a reconstruction model (by the wax-plate method of Born), *Otol.* 34; discussion (p. 35): Mr. S. R. Scott, Mr. Jenkins (reply)
 —, specimen of fracture of the temporal bone, *Otol.* 88
- Jensen's method (modified) in removal of ethmoidal epithelioma, *Laryng.* 66
- JEWESBURY, R. C.—Discussion on case for diagnosis, *Child.* 24
 —, infantile heart showing nodules on endocardium, *Child.* 45; discussion (p. 45): Dr. Parkinson, Dr. F. Parkes Weber, Dr. E. I. Spriggs
- JOHNSON, R., and LAWRENCE, T. W. P.—Retroperitoneal teratoma connected with the spinal canal, *Path.* 43
- Johnston, W., account of personal attack of diarrhoea due to laboratory examination of diarrhoeal dejecta, *Epid.* 97
- Joints, contracted, treatment by fibrolysin, *Clin.* 89, 90
- Jonas, H. C., precocious sexual development, quoted, *Path.* 55
- JONES, H. E.—Discussion on adventitious membrane, resembling normal drum, formed after radical mastoid operation, *Otol.* 59
 —, discussion on chronic middle-ear suppuration, *Otol.* 44
 — on thickening of the cartilage of both auricles (? othæmatoma), *Otol.* 53
- JONES, H. LEWIS.—Discussion on indications for electro-therapy in some gynaecological affections, *Obst.* 25
 —, discussion on therapeutical use of statical electricity, *Electr.* 83
 — on treatment of nævi by electricity, cautery, and refrigeration, *Electr.* 47
 — on vaccine therapy, *R.S.M.* 158
 —, treatment of nævi, *Electr.* 37, 38
- JONES, J. ARNOLD.—Mucous polypus presenting at the pharyngeal orifice of the left Eustachian tube in a man suffering from bilateral chronic adhesive otitis media, *Otol.* 55
- JONES, R. LLEWELYN, arthritis deformans, *Baln.* 85; discussion (pp. 95-98): Dr. F. Fox, Dr. P. King, Dr. Buckley, Dr. Hale White, Dr. T. F. Gardner, Mr. G. Watson, Dr. F. H. Humphris, Mr. Midelton, Dr. Solly, Dr. Ackerley, Dr. A. E. Garrod, Dr. P. W. Latham, Mr. Wm. Armstrong, Dr. Sunderland, Dr. Llewelyn Jones (reply)
- JORDAN, A. C.—Discussion on case of fracture of humerus at site of innocent cyst, *Clin.* 195
- Kangalugi root, botanical identification, *Therap.* 67
 —, report on (H. W. Gadd), *Therap.* 67
 —, therapeutical value doubtful, *Therap.* 68
- Kasr-el-Ainy Hospital, Cairo, mortality from tetanus at (1906-08), *Med.* 39, 54
- Kausch, statistics of operation for gastric cancer, with involvement of lymphatic glands, *Surg.* 147

- Kehr, on pancreatic reaction, quoted, *Med.* 181
- Keith, A., on fissure in mid-line of nose, quoted, *Laryng.* 85
- , pathological report on specimen of the face and mouth of a female infant, showing arrest of development of the right half of the tongue, combined with a cleft of the soft palate and a palato-lingual fold, *Laryng.* 99
- Kellas and Foulerton, A. G. R., bactericidal agency of nitric acid and ozone formed in brush discharges, *Electr.* 84
- Keloid (multiple), associated with "neurotic excoriations" of "dug-out" type of Colcott Fox (H. G. Adamson), *Derm.* 42
- Keloids, acne vulgaris with (E. G. G. Little), *Derm.* 107
- KELSON, W. H.—Case of paresis of palate, pharynx and œsophagus, *Laryng.* 90; discussion (p. 91): Dr. Dundas Grant, Dr. H. J. Davis, Dr. Watson Williams, Dr. Kelson (reply)
- , case of tuberculous disease of the epiglottis, *Laryng.* 112
- , discussion on adventitious membrane, resembling normal drum, formed after radical mastoid operation, *Otol.* 59
- on lupus of nose and throat, *Laryng.* 87
- , frontal sinus disease, with necrosis, in a man aged 33, *Laryng.* 28
- KENNEDY, R. F.—Discussion on cases of cerebral thrombosis, *Neur.* 86
- , discussion on syringomyelia with total anaesthesia, *Neur.* 103
- Kensington, diarrhoea in, fortnightly incidence compared with that of whole of London, *Epid.* 100
- , influence of aggregation on, *Epid.* 104
- , multiple cases, consecutive dates of onset, *Epid.* 102, 103
- in houses where deaths occurred, *Epid.* 102
- invasion of houses and families, date of onset of cases, *Epid.* 100, 101
- Keratinization absent in case of epithelioma of tongue, *Clin.* 180
- Keratitis, tuberculous and scrofulous, treatment by application of violet-flame discharge, *Electr.* 77
- Keratoderma, gonorrhœal (keratoderme blennorrhagique), case of (J. H. Sequeira), *Derm.* 77-80
- , palmar and plantar, symmetrical and congenital, of Besnier, case of (E. G. G. Little), *Derm.* 31
- Keratosis, refrigeration in treatment of, *Electr.* 44
- obturans, see *Cholesteatoma*
- palmaris et plantaris, associated with generalized ichthyosis (H. G. Adamson), *Derm.* 83
- KERR, J. M. MUNRO.—Case of placenta prævia, in which it was deemed advisable to perform Caesarean section, *Obst.* 11; discussion (p. 13): Sir William Smyly, Dr. W. S. A. Griffith, Dr. Herbert Williamson, Dr. Macnaughton-Jones, Dr. Munro Kerr (reply)
- , statistics of puerperal eclampsia, quoted, *Obst.* 201
- Kettlitz, Caesarean section for eclampsia, quoted, *Obst.* 196, 198
- Kidney, amount of urea excreted by, depends on amount of protein eaten, *Therap.* 141
- , diseases of, choice of anæsthetic for subjects of, *Anæsth.* 69
- , treatment by ether inhalation without admixture of exhalations from lungs (R. H. Hodgson), *Clin.* 213, 215
- , granular, comparison with osteo-arthritis, *Baln.* 102
- , pericardial effusion associated with, *Med.* 56, 97
- , pericarditis associated with, *Med.* 97, 103
- , radiographs produced through Snook's high-tension transformer, *Electr.* 158
- , tubules of, calcium oxalate found in, after experimental poisoning by oxalic acid, *Med.* 6, 7
- Kidneys, excretion of common salt from body by, *Baln.* 67, 68, 69
- , in excess, cause of renal inadequacy, *Baln.* 69, 72
- , dorsal percussion by (Piorry), *Med.* 217
- Killian's operation, performance on children, *Laryng.* 57, 58
- Kindl, recovery after palliative operation for supposed malignant disease of stomach, *Surg.* 138, 139

- KING, P.—Discussion on arthritis deformans, *Baln.* 95
 Kingsford, tuberculous infection of tonsils in children, quoted, *Child.* 30
 Kinnicutt, on pancreatic reaction, quoted, *Med.* 182
 KNAGGS, R. L.—Statistics of intussusception in children, *Child.* 172
 Knee-joint, tuberculosis of, recovery after treatment with tuberculin, R.S.M. 115
 Knee-joints (pyemic) treated by tapping and incision (E. M. Corner and M. Huggins), *Clin.* 120
 Kobert and Küssner, effects of experimental poisoning by oxalic acid, quoted, *Med.* 6, 7
 Koch, R., attitude of medical profession upon discovery of tuberculin by, R.S.M. 108
 —, oral administration of tuberculin stated to be inert by, R.S.M. 198
 Kocher, Th., cause of rupture of tunica vaginalis in hydroceles, quoted, *Surg.* 171
 —, on effects of cachexia strumipriva, quoted, *Path.* 106
 Kocher's method of gastrectomy, *Surg.* 140
 — of operation in malignant disease of tongue, *Clin.* 183
 Koplik, tuberculous infection of tonsils, quoted, *Child.* 29
 Koplik's spots in measles, *Epid.* 54
 Korányi, percussion values of spinous processes, *Med.* 214
 Korotkow, suggestion of method of combination of auscultatory and tactile methods of reading arterial pressure, *Med.* 210
 Krafft-Ebing, immunity to syphilitic re-infection, quoted, *Neur.* 46
 —, inoculation of general paralytics to prove immunity to syphilis, *Neur.* 48
 Krait (banded), see *Bungarus fasciatus*
 — (common), see *Bungarus caeruleus*
 Kraurosis vulvæ, definition, *Obst.* 35
 — —, pathology, *Obst.* 36
 — — and carcinoma vulvæ, leucoplakic vulvitis and its relation to (C. Berkeley and V. Bonney), *Obst.* 29
 — —, with commencing carcinoma (A. E. Giles), *Obst.* 54
 Kraus, hæmorrhage in duodenal ulcer, quoted, *Surg.* 74
 —, vaccine therapy of syphilis, quoted, *Surg.* 229
 Krause, distinction between inflammatory and malignant disease of stomach, *Surg.* 139
 Krause's method of induction of premature labour, *Obst.* 125
 Krebber, Dr., new tonsil instrument, *Laryng.* 134
 Kreuznach, cure of female sterility at, explained, *Baln.* 10
 Krienitz, on pancreatic reaction, quoted, *Med.* 182
 Kruckmann, tuberculous infection of tonsils, quoted, *Child.* 28
 Kuhn's instruments for per-oral intubation, demonstration of (W. Milligan), *Otol.* 49
 Kurth, on *Streptococcus conglomeratus*, quoted, *Otol.* 20
 Küssner and Kobert, effects of experimental poisoning by oxalic acid, quoted, *Med.* 6, 7
 Kutnow's powders, claims advanced as to, *Therap.* 86
- Laboratories, workers in, subserviency to clinicians, R.S.M. 78
 Laboratory reports on specimens, compared with cases under consultation between specialist and practitioner, R.S.M. 6, 7
 Laboratory tests, use of, cannot displace or dispense with physical diagnosis, R.S.M. 111
 Labour, character of, inseparable from health and physical state during pregnancy, *Obst.* 127
 —, obstructed by large cervical fibroid in vagina (W. S. A. Griffith), *Obst.* 188
 — (premature), induction of, in contracted pelvis, *Obst.* 115, 120, 121, 123
 — — — — —, Krause's method, *Obst.* 125
 — — — — —, results at Queen Charlotte's Hospital, *Obst.* 125
 —, spontaneous transverse rupture of uterus at fundus during (A. H. N. Lewers), *Obst.* 136
 Labour (industrial, casual), organization of, to secure continuous employment, *Epid.* 36
 — — —, decasualization of, *Epid.* 35, 36
 — — —, skilled and unskilled, training in, *Epid.* 38

- 6

- Larynx, stenosis of (cicatricial), case of, treatment (W. Hill), Laryng. 120
 ——— (complete), left after diphtheria and tracheotomy, completely cured by repeated intubations spread over three years (StC. Thomson), Laryng. 95
 ——— inspiratory, in man (StC. Thomson), Laryng. 112
 ———, with hypertrophic laryngitis in subject of syphilis (W. Hill), Laryng. 93
 ———, tuberculosis in, site of development, Laryng. 159, 160
 ——— of, in girl (G. C. Cathcart), Laryng. 127
 ———, extensive, in middle-aged man, treatment by galvano-cautery; recovery almost complete (J. Dundas Grant), Laryng. 87
 ———, with considerable dysphagia, treated and relieved by congestion hyperemia (W. Howarth), Laryng. 164
 ———, with extreme odynphagia, relieved by injection of alcohol into left superior, laryngeal nerve (J. Dundas Grant), Laryng. 153
 ——— (ventricle), specimens and case illustrating diseases of (W. Jobson Horne), Laryng. 157
 ——— (right), ? prolapse of, in chronic laryngitis (H. B. Robinson), Laryng. 30
 ——— and fauces, nasal cavities, tongue, cheek and lips, affected by pemphigus vegetans, Laryng. 78
 ——— and throat, variation in effect of "back" and "belly" breathing on stresses, strains and frictions in, illustrated by model (R. H. Scanes Spicer), Laryng. 41
 ——— and trachea, portions of, with specimen of goitre, from woman (J. Donelan), Laryng. 38
 ———, see also *Vertigo (laryngeal)*
- LATHAM, A.—Administration of vaccine by mouth, quoted, R.S.M. 37
 ———, correlation of temperature to opsonic index, quoted, R.S.M. 33, 41
 ———, discussion on vaccine therapy, R.S.M. 113
 ———, tuberculous infection of tonsils, quoted, Child. 30
 ———, and Inman, A. C., oral administration of horse serum, quoted, R.S.M. 199
- LATHAM, P. W.—Discussion on arthritis deformans, Baln. 103
- LAW, EDWARD.—Deafness and discomfort in the right ear as early symptoms in a case of epithelioma originating near the right Eustachian tube, Otol. 28; discussion (p. 29): Dr. Law, Dr. Paterson, Mr. H. Tilley, Dr. H. J. Davis, Mr. C. H. Fagge, Mr. A. L. Whitehead, Dr. Dan McKenzie, Mr. Westmacott, Dr. Bronner, Dr. Law (reply)
 ———, discussion on case of audible tinnitus, Otol. 2, 54
 ——— on exostosis of right external meatus, Otol. 77
 ——— on hysterical deafness with active vestibular reactions, Otol. 74
 ——— on well-defined Ménière's symptoms, Otol. 52
 ——— on objective clicking sound in left ear, Otol. 37
 ———, remarks on letter from Professor Politzer, Otol. 18
- LAWRENCE, L. A.—Case of ulceration of palate, Laryng. 164
 ———, congenital absence of the bony part of the palate in a girl aged 16, Laryng. 163; discussion (p. 163): Dr. J. Dundas Grant
- LAWRENCE, T. W. P., and JOHNSON, R.—Retropertitoneal teratoma connected with the spinal canal, Path. 43
- LAWSON, DAVID.—Discussion on vaccine therapy, R.S.M. 197
- LAWSON'S reaction, R.S.M. 94, 95
- LAZARUS-BARLOW, W. S.—Discussion on surgery of malignant disease of the tongue, Clin. 186
 ———, discussion on treatment by radium of malignant growths, Clin. 165
 ———, endothelioma of tongue, quoted, Clin. 179
- Lead neuritis, chromatolysis of Betz cells in, Path. 17
- Leathes, J. B., on fatty liver and heart, quoted, Child. 82
- Lebedeff, on fatty liver, quoted, Child. 82, 83
- LEDINGHAM, J. C. G.—Discussion on chloroma, Child. 97
 ——— and FIRTH, H. C. D.—Hemiatrophy of cerebral hemisphere, Child. 53
- Le Dran, cause of rupture of tunica vaginalis in hydroceles, quoted, Surg. 171
- LEE, C. G.—Discussion on case of audible tinnitus, Otol. 2
 ———, discussion on pathological changes found in ear of deaf-mutes, Otol. 14

- LEE, P. G.—Discussion on cases of gout, Clin. 24
- LEES, DAVID B.—Discussion on pericardial effusion, Med. 69
- , physical signs of incipient pulmonary tuberculosis and its treatment by continuous antiseptic inhalations, with results in thirty cases, Therap. 17; discussion (p. 35): Dr. Ewart, Dr. Duncanson, Professor Cushny, Dr. Lees (reply)
- , rheumatic pericarditis, alluded to, Med. 75
- Leg, congenital œdema of (M. Smith), Child. 89
- Leg tremor during anæsthetization, Anæsth. 68
- Legs, bending of bones of both, with partial ankylosis of both hips (S. Hastings), Clin. 95
- LEGGE, T. M.—Discussion on perforation of nasal septum from salt dust, Laryng. 4
- Legrand and Carriev, fibromyoma of tube, quoted, Obst. 239
- Leicester, diarrhœa in, diminution in prevalence, Epid. 111, 113
- , typhoid fever in, diminution in prevalence, Epid. 111, 113
- , water-closet system prevalent in, Epid. 111
- LEISHMAN, Sir W. B.—Antityphoid inoculation, quoted, R.S.M. 37
- , discussion on vaccine therapy, R.S.M. 80
- Lenhartz method in dietetic treatment of gastric ulcer, acute, Therap. 127, 137
- — — — —, chronic, Therap. 130, 131, 137
- LEON, J. T.—Case of syphilitic dermatitis, Child. 159; discussion (p. 160): Dr. Pernet
- , specimen of large-celled sarcoma of the brain, Child. 160
- — — — — of peritoneal growths from a case of lymphocythæmia with blood-slides, Child. 160
- Leonard, proportion of ureteral to renal calculus, quoted, Electr. 94, 95
- Leprosy, case of (J. M. H. MacLeod), Derm. 111
- Lepto-meningitis, diffuse basal, complicating middle-ear disease preceded by labyrinthitis, Otol. 26
- Leptothrix, presence in pyorrhœa alveolaris, R.S.M. 173
- LETT, HUGH.—Cyst of the humerus, fracture, operation, Child. 98; discussion (p. 99): Mr. Lockhart Mummery
- Leube's method in dietetic treatment of chronic gastric ulcer, Therap. 130, 131
- Leucocytes, action of blood-fluids on, method of studying (H. Chambers), Path. 73
- — — — — of micro-organisms on, method of studying, Path. 75
- Leucocytosis and purulent pericarditis, Med. 65, 66
- Leucoderma, congenital, Clin. 197
- syphiliticum (J. E. R. McDonagh), Derm. 114
- — — — — simulated by multiple scars (H. G. Adamson), Derm. 1
- Leucoma, definition of, Laryng. 88
- Leucorrhœa, cause of kraurosis vulvæ, Obst. 38
- and leucoplakic vulvitis, Obst. 39
- Leukæmia, action of Röntgen rays on, R.S.M. 158
- (medullary), following traumatic pancreatic cyst cured by operation after twelve years' interval (A. E. Barker), Clin. 121
- (myelogenous), in infancy, case of (T. R. Whipham), Child. 92
- — — — —, rarity of, Child. 93, 94
- , in infant aged 18 months, pathological report on case (T. R. Whipham), Child. 152
- (spleno-medullary), intercurrent erysipelas (J. Galloway), Clin. 195
- Leukoplakia (?) of soft palate (A. Wylie), Laryng. 82
- Levaditi, demonstration of presence of spirochete within ovum, quoted, Neur. 46
- Levisohn on pancreatic reaction, quoted, Med. 182
- LEVY, A. G.—Discussion on lymphatism, Anæsth. 51
- LEWERS, A. H. N.—Discussion on abdominal hysterectomy for necrosed and suppurating subperitoneal fibroid, Obst. 65
- , discussion on slighter forms of puerperal sepsis, Obst. 239
- , double uterus (atresia of one-half) with dysmenorrhœa, Obst. 241; discussion (p. 242): Dr. Griffith, Dr. Blacker, Mr. Alban Doran

- LEWERS, A. H. N.—Note on a case of cystic tumour of the right broad ligament, springing from the uterus, and apparently developed from Gartner's duct, *Obst.* 67; discussion (p. 70): Mr. Doran, Dr. Routh, Dr. Maxwell, Mrs. Boyd, Dr. Lewers (reply)
- , spontaneous transverse rupture of the uterus at the fundus during labour, *Obst.* 136; discussion (p. 138): Dr. R. Drummond Maxwell
- Leyden jars, use of, in therapeutic applications of static electricity, *Electr.* 80
- Lichen planus associated with diabetes, case of (J. Galloway and A. W. Williams), *Derm.* 45
- of anomalous aspect and course (E. G. G. Little), *Derm.* 107
- in child (J. E. R. McDonagh for P. S. Abraham), *Derm.* 89
- case of (H. Davis), *Derm.* 104
- annularis, case of (H. G. Adamson), *Derm.* 67
- hypertrophicus, case for diagnosis (E. G. G. Little), *Derm.* 87
- , in boy (S. E. Dore), *Derm.* 16
- scrofulosorum (E. G. G. Little), *Derm.* 2
- simplex chronicus of Vidal, case of (H. G. Adamson), *Derm.* 41
- Lieven, W. A., on treatment of syphilis, quoted, *Surg.* 245
- Ligament, right broad, cystic tumour of, springing from uterus, and apparently developed from Gartner's duct (A. H. N. Lewers), *Obst.* 67
- Limb, congenital enlargement of one, occurring in brother and sister (O. L. Addison), *Child.* 43
- Limbs, shortness of, in animals, relation to thyroid deficiency, *Path.* 107
- Linser, serum reaction in children of syphilitic patients, quoted, *Neur.* 120
- Lip (lower), inside of cheek, and tongue, syphilitic ulceration (C. Nourse), *Laryng.* 68
- (upper), beard region and hand, ringworm affecting, in man aged 28 (S. E. Dore), *Derm.* 111
- Lipidol in treatment of syphilis, *Surg.* 236
- Lipochromes, *Neur.* 48
- Lipoclastic accelerating action of serum as an index in pathological conditions (J. A. Shaw-Mackenzie and O. Rosenheim), *Path.* 168
- Lips, unusual condition of (Sir M. Morris and S. E. Dore), *Derm.* 18
- , tongue, cheek, larynx, fauces and nasal cavities affected by pomphigus vegetans (H. J. Davis), *Laryng.* 78
- Lipoid substances, definition of, *Neur.* 48
- , groups of, *Neur.* 48
- Liquid air, difficulties in treatment by, *Electr.* 42
- Lithotomy, see *Calculus, removal*
- LITTLE, E. G. GRAHAM.—Acne vulgaris with keloids, *Derm.* 107
- , acute and very extensive eruption of erythema iris type in a man aged about 35, *Derm.* 2
- , case for diagnosis, *Derm.* 17, 29; discussion (p. 18): Dr. Colcott Fox
- of kératodermie palmaire et plantaire symétrique et congénitale of Besnier, *Derm.* 31
- of lichen scrofulosorum, *Derm.* 2
- of Recklinghausen's disease, *Derm.* 49
- , discussion on case for diagnosis, *Derm.* 14
- of rodent ulcer, *Derm.* 5
- , disseminated lupus in a girl aged 4½, *Derm.* 59; discussion (p. 59): Dr. Colcott Fox
- , lichen planus of anomalous aspect and course, *Derm.* 107
- , lupus erythematosus with symptoms indicating the possibility of the transformation of the circumscribed type into that of the acute disseminated form, *Derm.* 18; discussion (p. 18): Dr. Colcott Fox
- , pigmented moles in a female child aged 2, *Derm.* 30
- , scleroderma of the circumscribed variety with unusually numerous patches, *Derm.* 48
- of the diffuse type in a woman aged 58, *Derm.* 47; discussion (p. 48): Dr. Stowers
- , two cases for diagnosis, *Derm.* 87

- LITTLE, E. G. GRAHAM.—Two cases for diagnosis, *Derm.* 70; discussion (p. 70): Dr. Whitfield, and (p. 72): Dr. H. G. Adamson, Dr. Pringle, Dr. Whitfield
 —, urticaria pigmentosa of very exceptional severity, *Derm.* 3
 —, and SEQUEIRA, J. H.—Case of pseudo-xanthoma elasticum, *Derm.* 69; discussion (p. 70): Dr. T. C. Fox
 Little, Gordon, solubility of urates, quoted, *Baln.* 73, 80
 Liver, abscess of, bursting upward and causing abscess in lung, *Surg.* 67
 —, treatment by vaccine inoculation, *R.S.M.* 57
 —, adenoma of, amyloid degeneration in (S. G. Shattock), *Path.* 153
 —, malignant (F. Parkes Weber), *Path.* 147
 —, atrophy, subacute, case of (A. D. Fordyce), *Child.* 1
 —, relation to cirrhosis, *Child.* 12, 13
 —, bile-producing primary malignant tumour of (F. Parkes Weber), *Path.* 147
 —, cancer, primary, of, *Path.* 150
 —, cirrhosis of, case (R. Hutchison), *Child.* 40
 —, and chronic pancreatitis, *Med.* 174
 —, connected with chronic ascites, omentopexy and peritoneal drainage for; patient shown a few months after (F. Parkes Weber), *Clin.* 80
 —, relation to subacute atrophy, *Child.* 12, 13
 —, disease of, causing persistent vomiting, *Child.* 1, 6, 10
 —, dorsal percussion of (Piorry), *Med.* 216
 —, fatty degeneration in acute starvation, *Child.* 82
 —, in phosphorus poisoning, *Therap.* 66
 —, in recurrent or cyclic vomiting, *Child.* 83
 —, great enlargement of, in case of rheumatoid arthritis with albuminuria (W. P. Herringham), *Clin.* 61
 —, hydatid cyst of, painless, *Obst.* 5
 —, lymphosarcoma, case of (A. D. Fordyce), *Child.* 1, 7
 —, malpositions of, cause, *Med.* 235
 —, microscopical section from, in case of icterus in infant (G. Carpenter), *Child.* 48
 —, and pancreas, cirrhosis of, in case of bronzed diabetes (H. S. French), *Path.* 94
 —, pancreas and skin, pigmentary deposits in, in case of bronzed diabetes (H. S. French), *Path.* 94
 —, sluggishness of, treatment by static wave current, *Electr.* 107
 Llaudrindod Wells, saline waters of, method of giving, *Baln.* 79
 Local reaction in vaccine therapy, *R.S.M.* 192
 Lochial discharge, foul-smelling in slight puerperal sepsis, *Obst.* 224
 Lockemann, organic disease, cause of olfactory discharges, quoted, *Neur.* 2
 LOCKYER, C.—Discussion on abscess of ovary, probably due to infection by the *Amœba coli*, *Obst.* 153
 —, discussion on atypical malignant tumour of uterus, *Obst.* 100
 —, on case of adenoma malignum cysticum cervicis uteri, *Obst.* 103
 —, of tubal mole, *Obst.* 237
 —, on fibro-adenoma of ovary, *Obst.* 52
 —, pelvic hæmatocele of ovarian origin, *Obst.* 106; discussion (p. 111): Dr. Macnaughton-Jones, Dr. Arthur Giles, Dr. Blacker (reply)
 —, report on specimen of myomatous tumour of uterus shown by Dr. H. Macnaughton-Jones, *Obst.* 104
 Locomotor ataxy, relief of, by application of static electricity, *Electr.* 84
 —, treatment by static wave current, *Electr.* 103
 London, diarrhoea in, incidence: fortnightly incidence in Kensington compared with, *Epid.* 100
 —, prevalence stationary, *Epid.* 112, 118, 119
 —, seasonal prevalence of scarlet fever in, date, *Epid.* 75
 —, typhoid fever in, diminution in prevalence, *Epid.* 112, 114, 115, 116, 118
 —, water-closet system established in, *Epid.* 111, 112

- Lupus, treatment by static electricity under spark application, Electr. 78-80, 84
 ————, cause of curative effect, Electr. 79, 84
 ———— by tuberculin, R.S.M. 105
 ———— injections, cases (G. T. Western), Derm. 8
 ———— by vaccine, results, R.S.M. 147
 ———— disseminatus, case of (E. G. Graham Little), Derm. 59
 ———— faciei (J. H. Sequeira), Derm. 61
 ———— erythematous almost exclusively limited to scalp, case of (Sir M. Morris), Derm. 98
 ———— associated with erythema iris (J. H. Sequeira), Derm. 94
 ————, case of (Sir M. Morris and W. Fox), Derm. 99
 ———— of eleven years' duration localized to scalp (J. H. Sequeira), Derm. 101
 ————, refrigeration in treatment of, Electr. 44
 ————, symptoms of, possible transformation of circumscribed into acute disseminated form (E. G. Graham Little), Derm. 18
 ———— unusual case (J. H. Sequeira), Derm. 62
 ————, with epithelioma (J. H. Sequeira), Derm. 100
 ———— with sclerodactyly (J. H. Sequeira), Derm. 102
 ———— of nose, hard palate, fauces and epiglottis (W. Stuart-Low), Laryng. 86
 ———— and throat, method of infection, Laryng. 87
 ————, treatment, discussed, Laryng. 87
 ————, treatment by tuberculin inoculation, results, R.S.M. 51, 52
 ———— vulgaris erythematodes, case of (S. E. Dore), Derm. 97
- Luxor as a health resort, Baln. 123
- Lying-in hospitals, German, puerperal morbidity in, Obst. 222
- Lymphadenoma, case of, starting at twenty-fifth month (R. Miller), Child. 135
 ————, diagnosis of enlarged, from tuberculous glands necessary in, Electr. 126
 ————, disappearance of enlarged glands in, under X-ray treatment (A. H. Pirie), Electr. 125-131
- Lymphangioma, association of capillary hamangioma (port-wine naevus) with, Clin. 112
 ———— of tongue, cases of (E. Rock Carling), Clin. 111 (A. E. Barker), Clin. 112
 ————, treatment, operative, Clin. 112
- Lymphatic area, removal of, in operations for cancer of stomach, Surg. 147-152
 ———— glands, condition, association with lymphatism, Anæsth. 52
 ————, examination in cases of lymphatism, Anæsth. 52
 ———— of neck, tuberculosis of, special site of invasion, Child. 31
 ———— and spleen, enlargement of, in rheumatoid arthritis (E. I. Spriggs), Clin. 15
 ———— permeation, path of, Surg. 147
- Lymphatics (intestinal) dilatation in case of congenital œdema (D. Forsyth), Child. 51
- Lymphatism (H. Bellamy Gardner), Anæsth. 19-36
 ————, ætiology, Anæsth. 30, 37
 ————, appointment of committee on, Anæsth. 64
 ————, association with acute nephritis, Anæsth. 55
 ———— with adenoids, Anæsth. 44
 ———— with enlarged thymus gland, cases illustrating, Anæsth. 21-30
 ———— with epilepsy, Anæsth. 53, 56
 ———— with rickets, Anæsth. 50, 53
 ———— with sterility, Anæsth. 60
 ————, causes of death, Anæsth. 49, 50
 ————, choice of anæsthetic for subjects of, Anæsth. 71
 ————, chronic, Anæsth. 56
 ————, condition of thymus gland in, Anæsth. 51
 ————, death under anæsthesia in cases of, Anæsth. 23-30, 32, 41, 42, 59
 ————, diagnosis, Anæsth. 38
 ————, physical signs of, Anæsth. 52, 53
 ———— and exophthalmic goitre, case of, operation under ether, Anæsth. 49
 ————, failure of recognition, Anæsth. 54

- Lymphatism, general toxæmia in, Anæsth. 57**
 — in relation to myasthenia gravis, Anæsth. 46-48
 — — — — —, anatomical changes, Anæsth. 47
 — — — — —, mode of death, Anæsth. 47
 — — — — — to thyroid-gland incompetency, Anæsth. 43
 — lack of knowledge of condition prevalent, Anæsth. 3
 —, pathological report on case (R. S. Trevor), Anæsth. 27
 —, pathology of, Therap. 39, 40
 —, previous administration of thyroid-gland extract in, Anæsth. 58
 —, prognosis in, Anæsth. 57
 —, quasi-acute, Anæsth. 56
- Lymphocythæmia, specimen of peritoneal growths from case of, with blood-slides (J. T. Leon), Child. 160**
- Lymphocytosis of cerebrospinal fluid in general paralysis and tabes, Neur. 40-42**
 — — — — — in secondary syphilis, Med. 36, 37
 — — — — — in syphilitic and parasyphilitic meningitis, diagnostic value, Neur. 39
- Lymphodermis perniciosa, Derm. 40**
- Lymphoid swelling and catarrhal jaundice, Med. 171**
- Lymphosarcoma of liver, case of (A. D. Fordyce), Child. 1, 7**
 — of tonsil removed by lateral pharyngotomy after Vohsen's method (G. Wilkinson), Laryng. 86
- Lymphosarcomatosis, specimens from case, with clinical and post-mortem notes (F. W. Higgs), Child. 17**
- LYSTER, C. R. C.—Discussion on pericardial effusion, Med. 94**
 —, discussion on treatment of malignant growths by radium, Clin. 164
- McBRIDE, P.—Discussion on case of endothelioma of temporal bone, Otol. 33**
 —, discussion on objective clicking sound in left ear, Otol. 37
- McCANN, FREDERICK JOHN.—Caesarean section in the treatment of eclampsia gravidarum, with notes of a successful case, Obst. 193-202; discussion (p. 203): Dr. Herman, Dr. Herbert Spencer, Dr. Amand Routh, Mr. W. D. Spanton, Dr. W. J. Gow, Dr. Macnaughton-Jones, Dr. McCann (reply)**
 —, case of tubo-abdominal pregnancy, Obst. 149
 —, degenerating fibromyoma of the cervix, Obst. 147
 —, discussion on carcinoma of prolapsed uterus, Obst. 163
 — — — — — on case of myomectomy during pregnancy, Obst. 166
- McCardie, W. J., status lymphaticus in relation to general anæsthesia, quoted, Anæsth. 19**
- Maccarty and Wilson, relation between ulcer and cancer of stomach from histological point of view, quoted, Surg. 137**
- McClean, J., local obesity of eunuchs, quoted, Path. 122**
- McCRIBBICK, T., and ROLLESTON, J. D.—Case of purpura fulminans, Child. 75**
- McCULLOCH, H. D.—Discussion on pancreatic reaction and fecal analysis, Med. 204**
 —, discussion on radium in treatment of malignant growths, Electr. 70
 — — — — — on treatment of nævi by electricity, cautery, and refrigeration, Electr. 49
 — — — — — on X-ray treatment of lymphadenoma, Electr. 136
- MCDONAGH, J. E. R.—Case of alopecia areata (exhibited for P. S. Abraham), Derm. 55**
 —, case of dermatitis herpetiformis, Derm. 89; discussion (p. 90): Sir Malcolm Morris, Dr. Pringle
 — — — — — of leucoderma syphiliticum, Derm. 114
 — — — — — of a mixed tumour (tricho-epithelioma papulosum and syringo-cystadenoma), Derm. 32
 — — — — — of phlebitis nodularis syphilitica, Derm. 72
 — — — — — of syphilitic erythema nodosum, Derm. 50; discussion (p. 51): Dr. Whitfield, Dr. MacLeod, Mr. McDonagh (reply)

- McDONAGH, J. E. R.—Cases of syphilis illustrating the importance of the Wassermann reaction, Derm. 90; discussion (p. 92): Dr. Pernet, Sir Malcolm Morris, Dr. Whitfield
 —, discussion on case of mycosis fungoides, Derm. 40
 — on present position and treatment of syphilis, Surg. 228
 — lichen planus in a child (for P. S. Abraham), Derm. 89
 —, specimen of *Spirochæta pallida*, prepared with indian ink, with demonstration of method, Derm. 73
 — and POTTER, COUBRO.—Chancre of the tongue, Derm. 51
 — and SMITH, E. ARCHIBALD.—Case of pachydermatocoele, Clin. 76
 McGregor, statistics of intussusception in children, Child. 171
 Machinery, development of, beneficial effects on national prosperity and health in Great Britain, Epid. 10
 McKENZIE, DAN.—Case of asthma; improvement after nasal treatment, Laryng. 116; discussion (pp. 116-117): Dr. Scanes Spicer, Dr. Pegler, Mr. Clayton Fox, Dr. H. J. Davis, Dr. J. Dundas Grant, Dr. McKenzie (reply)
 — case of bilateral abductor paralysis from central nerve disease, Laryng. 162; discussion (p. 162): Mr. C. Fox
 — of tabetic deafness, Otol. 38; discussion (p. 39): Mr. Wilkinson, Dr. McKenzie (reply)
 — with well-defined and transitory Ménière's symptoms; ? migraine with auditory and vestibular phenomena, Otol. 50; discussion (p. 52): Dr. Dundas Grant, Dr. E. Law, Dr. Pritchard, Dr. McKenzie (reply)
 —, congenital occlusion of the left posterior nares in a girl aged 19, Laryng. 91; discussion (p. 91): Dr. Paterson, Mr. Barwell, Dr. H. J. Davis, Dr. McKenzie (reply)
 —, discussion on case of laryngeal paralysis, Laryng. 151
 — on early symptoms in case of epithelioma originating near right Eustachian tube, Otol. 31
 — on endothelioma of ethmoid, Laryng. 68
 — on excision of entire tongue, Laryng. 139
 — on exostosis of right external meatus, Otol. 78
 — on pathology of cholesteatomata, Otol. 70
 — on peculiar erythema of left palate in a man aged 41 who had been a considerable smoker, Laryng. 27
 — on post-operative tests in labyrinthine disease, Otol. 6
 — on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 58
 — on rare congenital deformity of nose, Laryng. 85
 — on symptoms simulating cerebellar abscess, Otol. 80
 — on treatment of tuberculous laryngitis by galvano-puncture, Laryng. 122
 — on vulsellum-catch forceps, Laryng. 126
 —, general infiltration of larynx, probably luetic, Laryng. 70; discussion (p. 71): Dr. Dundas Grant
 —, perforation of nasal septum from salt (NaCl) dust, Laryng. 3; discussion (p. 4): Dr. Grant, Mr. de Santi, Dr. Legge, Dr. H. J. Davis, Mr. Clayton Fox, Mr. Rose, Dr. McKenzie (reply)
 —, thickening of the cartilage of both auricles (? othematoma), Otol. 53; discussion (p. 53) Dr. Milligan, Mr. Hugh E. Jones, Dr. McKenzie (reply)
 — and ABERCHROMBIE, P. H.—Hysterical deafness, with active vestibular reactions, Otol. 74; discussion (p. 75): Dr. Law, Mr. Scott, Dr. McKenzie (reply)
 — and GRANT, J. DUNDAS.—Epithelioma of left vocal cord in a woman aged 58, removal by thyrotomy, Laryng. 19
 Mackenzie, H. W. G., on danger of oral sepsis, quoted, Odont. 101, 102
 MACKENZIE, J.—Discussion on treatment of endocarditis, Therap. 113
 McKERRON, R., on contracted pelvis, quoted, Obst. 127
 MACLEOD, J. M. H.—Case for diagnosis, Derm. 112

- MACLEOD, J. M. H.—Case of cystic rodent ulcer, Derm. 3; discussion (p. 5): Sir Malcolm Morris, Dr. Little, Dr. Sequeira
 —, case of leprosy, Derm. 111
 — of syphilitic erythema nodosum, Derm. 51
 — of syphilitic onychia, Derm. 59
 — of xanthoma tuberosum multiplex, Derm. 108
 —, discussion on case for diagnosis, Derm. 14, 94
 — of lichen planus annularis, Derm. 67
 — of onychia sicca syphilitica, Derm. 44
 — on elephantiasis, associated with tertiary syphilis, Derm. 83
- McMULLAN, G.—Case of adiposis dolorosa, Clin. 55; discussion (p. 58): Mr. Pearce Gould, Dr. Gossage, Mr. Warren Low, Dr. Leonard Williams, Dr. Poynton, Sir Dyce Duckworth, Dr. Galloway, Dr. F. Parkes Weber, Dr. G. McMullan (reply)
- MACNALT, A. S.—Case of thoracic aneurysm in a boy, Clin. 35; discussion (p. 36): Dr. Parkes Weber, Dr. Herringham, Dr. Rolleston, Dr. Morley Fletcher, Dr. N. Dalton, Dr. MacNalty (reply)
- MACNAUGHTON-JONES, H.—An acoustic aid for persons partially deaf from various causes, Otol. 40
 —, discussion on abdominal hysterectomy for necrosed and suppurating subperitoneal fibroid, Obst. 66
 — on adenofibromatous polypus of uterus, Obst. 157
 — on adenomyoma of Fallopian tube, Obst. 240
 — on apparatus for continuous proctoclysis, Obst. 81
 — on atypical malignant tumour of uterus, Obst. 100
 — on Caesarean section in eclampsia gravidarum, Obst. 206
 — in placenta prævia, Obst. 14, 180
 — on carcinoma of prolapsed uterus, Obst. 163
 — on case of bilateral tuberculous pyosalpinx, Obst. 81
 — of primary carcinoma of Fallopian tube, Obst. 78
 — on condition of mouth in chronic disease, Odont. 104
 — on electro-therapy in some gynecological affections, Obst. 27
 — on extensive retroperitoneal hæmatocele complicating large uterine fibroid, Obst. 142
 — on fibro-adenoma of ovary, Obst. 53
 — on leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, Obst. 48, 50
 — on life-history of fibromyomata of uterus, Obst. 220
 — on malignant and innocent ovarian growths, Obst. 96, 97
 — on microscopic appearances in tubal pregnancy, Obst. 161
 — on modern methods of delivery in contracted pelvis, Obst. 126
 — on pelvic hæmatocele of ovarian origin, Obst. 111
 — on slighter forms of puerperal sepsis, Obst. 233, 234
 — on specimen from case of carcinoma of ovary, Obst. 72
 — of torsion of body of uterus, Obst. 118
 — on very large cervical fibroid, Obst. 185
 —, presidential address: pain associated with disorders of the female genital organs, Obst. 1-10; discussion (p. 10): Sir F. H. Champneys, Mr. W. D. Spanton
 —, myomatous tumour of the uterus simulating sarcomatous growth, Obst. 103
 —, report on specimen by Dr. Cuthbert Lockyer, Obst. 104
- MACWATERS, J. C.—Discussion on vaccine therapy, R.S.M. 172
 —, treatment of glycosuria by staphylococcus vaccine, quoted, R.S.M. 18
- McWilliam, delirium cordis of, Anæsth. 1, 5
- Madder, feeding with, effect on bones and teeth of pigs, Odont. 80, 83
- "Magenblase" and bismuth meals, Electr. 111
- Magnesium-sulphate solution, effects on injection into nerve trunks, Med. 41, 53
 — in treatment of tetanus, combined with antitoxin, Med. 41

- Magnesium-sulphate solution, effects in treatment of tetanus by intraspinal injection (L. Phillips), *Med.* 39
- — — — —, details and résumé of cases, *Med.* 43, 46, 51
- — — — —, risks of, *Med.* 45, 46, 52
- — — — —, technique, *Med.* 42
- — — — —, value determined by experiments on monkeys, *Med.* 41
- — — — — by subcutaneous infection, *Med.* 50
- MAGUIRE, ROBERT.—On oxaluria and the treatment of calcium-oxalate deposit from the urine, with a method for the solution of calcium-oxalate calculus whilst in the urinary passages, *Med.* 1-20; discussion (p. 21): Dr. Robert Hutchison, Dr. Beckett Overy, Dr. Nestor Tirard, Dr. Maguire (reply)
- MAHOMED, G.—Atmospheric electricity, *Baln.* 49-58
- Mahommedan, case of gout with large tophaceous deposits in (Sir D. Duckworth), *Clin.* 21
- Mahommedans, gout rare among, *Clin.* 22
- Mailhouse, fatal case of myasthenia gravis, quoted, *Anæsth.* 47
- MAIR, L. DARRA.—Discussion on summer diarrhoea and enteric fever, *Epid.* 270
- Makins, G. H., lateral compared with end-to-end anastomosis, quoted, *Surg.* 141
- Makkas, comparative mortality of cases of gastric cancer involving other organs, *Surg.* 145, 146
- — — — —, presence of emaciation in gastric cancer, quoted, *Surg.* 133
- MALCOLM, J. D.—Cholecyst-duodenostomy for acute emaciation, following the formation of a biliary fistula, *Clin.* 83; discussion (p. 85): Mr. A. Pearce Gould, Mr. Carless, Mr. Malcolm (reply)
- Males, greater prevalence of typhoid fever among, *Epid.* 184
- Malignancy, low grade of, possessed by tumours of pineal body, *Neur.* 71
- Malignant cerebral tumour, special sense discharge from, *Neur.* 2, 8
- — — — — disease, complicating chronic pancreatitis, *Med.* 169
- — — — —, growths in nasopharynx resembling adenoids, suggesting (J. L. Dundas Grant), *Laryng.* 72, 73
- — — — —, invasion of pancreas by growths of, from neighbouring organs, *Med.* 175
- — — — —, metastatic deposits of growth from, in pancreas, *Med.* 175
- — — — — of neck, undergoing treatment by radium (W. Hill), *Laryng.* 25
- — — — — of pancreas, pancreatic reaction in cases of, *Med.* 178
- — — — — of tongue, surgery of, and notes of three rare tumours on base of tongue (F. Eve), *Clin.* 173
- — — — — (probable) of larynx in woman (H. Barwell), *Laryng.* 36
- — — — —, resection of bladder for, case of (J. W. Thomson Walker), *Clin.* 68
- — — — — simulated by gumma of breast (H. J. Paterson), *Clin.* 82
- — — — —, squamous-celled, of larynx, parts of pharynx and œsophagus, excision (A. Evans), *Clin.* 44
- — — — —, treatment by mixed toxins, *Therap.* 79
- — — — — by radium, *Electr.* 69, 70
- — — — — growth (probably), laryngeal (H. W. FitzGerald Powell), *Laryng.* 74
- — — — —, of larynx, extrinsic (P. Watson Williams), *Laryng.* 145
- — — — —, slow growing, case for diagnosis (P. Watson Williams), *Laryng.* 60
- — — — — in neck, treated with radium (N. S. Finzi and W. Hill), *Clin.* 159
- — — — — of right temporal bone, with extension through external meatus, resembling aural polypus (P. W. Williams), *Otol.* 85
- — — — —, secondary, affecting spinal roots, case of (E. Farquhar Buzzard), *Neur.* 124
- — — — —, — — — — —, microscopic examination necessary to determine presence of, *Neur.* 125, 127-129
- — — — —, pathological report (C. M. Hinds Howell), *Neur.* 125
- — — — — stricture below large globular dilatation of upper third of œsophagus (W. Hill), *Laryng.* 165
- — — — — tumours, inoperable, curative effect of accidental erysipelas in, *Surg.* 2, 9
- — — — — of jaws, diagnosis by X-rays, *Odont.* 158, 161

- Malignant tumours, microbic origin of, presumed, Surg. 10
 ———, primary, of liver ("malignant adenoma") (F. Parkes Weber), Path. 147
 ———, question of retention of some metabolic or functional characteristics of original cell (F. Parkes Weber), Path. 147, 151
 ———, trauma or injury a factor in causation of, how explained, Surg. 10, 11
- MALINS, E.—Discussion on slighter forms of puerperal sepsis, Obst. 232
- Malnutrition in children benefited by lactic-acid administration, Therap. 54, 55
 ——— in infancy, connexion of hypoplasia of enamel with, Obst. 79, 82, 84
- MALONEY, W. J.—Case of cerebellar tumour, Neur. 99
 ——— (for Dr. J. Taylor), case of disseminated sclerosis, Neur. 99
- Malonic acid, Med. 6
- Malta, diarrhoea in, greatest prevalence in rural districts, Epid. 104, 105, 111, 118, 119
- Man, aged 42, ateliosis in, physical development arrested at age of 9 years (F. Parkes Weber), Child. 143
- Manchester, causes of deaths occurring in public hospitals, Epid. 18, 19
 ———, common lodging-houses in, Epid. 26
 ———, focus of infection for phthisis, Epid. 27
 ———, diarrhoea, death-rates (1891-1906), Epid. 176, 177
 ——— in, deductions from fatal cases among infants, Epid. 103, 104: see also under *Summer diarrhoea*, below
 ———, house-flies caught in, compared with deaths from diarrhoea, Epid. 148-149
 ———, prevalence of, in, correction of curves, Epid. 170-174
 ———, incidence of scarlet fever in, Epid. 90, 91
 ———, milk clauses (1899), administration of, Epid. 220
 ———, milk supply, inspection of, and results, Epid. 222-225
 ———, percentages of deaths occurring in public hospitals, Epid. 18
 ———, phthisis in, classes of labour in which death-rate is high, Epid. 29, 30
 ———, poverty in, method of estimation, Epid. 13
 ———, summer diarrhoea in, Epid. 134-136
 ——— in 1904-1909, deaths from, Epid. 148-152
 ———, death-rate compared with temperature and rainfall, Epid. 167, 168
 ———, deaths from, compared with onset of typhoid-fever cases (1891-1906), Epid. 192-206
 ———, fatal cases in, relation to prevalence of flies, Epid. 169
 ———, relation of privy-midden system to, Epid. 167, 168
 ———, typhoid fever in, causes of prevalence, Epid. 186, 187
 ———, death-rates (1891-1906), Epid. 178, 179
 ———, influence of consumption of shell-fish on, Epid. 190, 191, 207-9
 ———, in relation to prevalence of flies, Epid. 175, 178, 179
 ———, transmission by house-flies, Epid. 189
 ———, vital statistics of (1901-1903), Epid. 17
 ———, relation of poverty to disease as deduced from (1901-1903), Epid. 17
 ——— (North), nature of population, natural elevation and sanitary districts, Epid. 16, 17
 ———, phthisis in, death-rates, males (1901-1905), Epid. 22, 23
 ———, females (1901-1905), Epid. 24, 25
 ———, total cases notified (1907), Epid. 20
 ——— (South), nature of population, natural elevation and sanitary districts, Epid. 16, 17
 ———, phthisis in, total cases notified (1907), Epid. 20
 ———, death-rates, males (1901-1905), Epid. 22, 23
 ———, females (1901-1905), Epid. 24, 25, 31
 ——— (Township), nature of population, natural elevation and sanitary districts, Epid. 16, 17
 ———, phthisis in, death-rates, males (1901-1905), Epid. 22, 23
 ———, females (1901-1905), Epid. 24, 25, 31
 ———, total cases notified (1907), Epid. 20
- MANDERS, H.—Discussion on lymphatism, notes on recent case, Anaesth. 42

- MANDERS, H.—Discussion on static wave current, *Electr.* 104
 —, discussion on treatment of naevi by electricity, cautery, and refrigeration, *Electr.* 48, 50
 Mandible, exostoses of (F. Coleman), *Odont.* 21
 —, fracture of, set with a silver splint made by the casting process (C. Schelling), *Odont.* 51
 — and canine tooth vertically fractured (W. de C. Prideaux), *Odont.* 92
 Mangiagalli, veratrum in treatment of puerperal eclampsia, *Obst.* 205
 Mann, J. Dixon, on behaviour of retained chlorides, quoted, *Baln.* 68
 Manometer (compressed air mercurial), use of, in reading arterial pressure by auscultatory method, *Med.* 210
 — and armlet, use of, in measuring arterial blood-pressure, *Med.* 208
 MANUEL, A.—Case of (?) congenital aortic stenosis, *Child.* 73
 —, pseudo-hypertrophic muscular paralysis, *Child.* 72
 —, two cases of acute anterior poliomyelitis involving the four limbs, *Child.* 21
 Marie and Bernard, cases illustrating effects of syphilitic virus on nervous system, quoted, *Neur.* 43
 Marienbad tablets, *Therap.* 86
 Marinesco, on axolysis, quoted, *Path.* 25
 —, perinuclear chromatolysis, quoted, *Path.* 16, 17
 —, regeneration of sciatic nerve after section, *Path.* 23
 Marnoch, statistics of intussusception in children, quoted, *Child.* 170
 Marriage unions, improvident and unhealthy, prevention among working classes, *Epid.* 43
 MARRIS, H. F., and GOSSAGE, A. M.—Statistics of pericardial effusion from Westminster Hospital, *Med.* 110
 MARSHALL, C. F.—Discussion on present position and treatment of syphilis, *Surg.* 233
 Marshall, J. S., on absorption of roots of permanent teeth, quoted, *Odont.* 15
 Martigny, waters, diuretic action explained, *Baln.* 9
 Martin, Professor A., statistics of ovarian growths, quoted, *Obst.* 97
 MARTIN, C. F.—Discussion on bluish pigmentation of skin, *Clin.* 141
 MARTIN, C. J., F.R.S.—Discussion on bovine tuberculous mastitis, *Epid.* 244
 Martin, S. H. C., F.R.S., channels of tuberculous infection, quoted, *Child.* 27
 MARTIN, W.—Discussion on proprietary, patent and secret remedies, *Therap.* 95
 Massage in heart failure during anaesthesia (V. B. Orr), *Anæsth.* 1
 —, sub-diaphragmatic, in cases of heart failure, *Anæsth.* 6, 8, 9
 Massol's bacillus, see *Bacillus bulgaricus* (lactic-acid bacillus)
 Massolettes, chocolate cream, *Therap.* 54, 59
 Mastication of food, *Odont.* 2, 3
 —, importance of inquiry as to methods of, *Odont.* 95, 96
 Mastitis (bovine tuberculous), cows in state of advanced tuberculosis chief factor in prevalence of, *Epid.* 239, 242, 255, 256
 — — —, data from examination of milk samples and inspection of cows and sheds, *Epid.* 220-225
 — — —, in relation to tuberculosis of other organs, *Epid.* 219, 220
 — — —, influence of altitude and rivers, *Epid.* 229
 — — —, of cowshed and herd, *Epid.* 231
 — — —, prevalence of (S. Delépine), *Epid.* 217-256
 — — —, influences determining (S. Delépine), *Epid.* 217-56
 Mastoid antrum, microscopical sections through, in fatal case of scarlet fever demonstrating *Streptococcus conglomeratus* in situ (R. Scott), *Otol.* 18
 — operation (double), deformity of both pinnae resulting from perichondritis following (H. J. Davis), *Otol.* 71
 — region, case of sarcoma of; radium treatment (N. S. Finzi), *Clin.* 157
 Matas's operation for popliteal aneurysm (Maynard Smith), *Clin.* 130
 — — — in both legs (H. M. Rigby), *Clin.* 131
 Maucclair, experiments on power of resistance of tunica vaginalis, quoted, *Surg.* 167
 Maxilla, suppurative of, two cases (C. Schelling), *Odont.* 113

- Maxilla, excision of both, for epithelial odontoma (A. Carless), Clin. 10
 — (superior), excision of both; extension of tumour to frontal sinus (A. Carless), Clin. 114
 Maxillary sinus, suppuration in, complicated by extensive fracture of nasal septum (J. Donelan), Laryng. 89
 MAXWELL, R. DRUMMOND.—Discussion on case of cystic tumour of right broad ligament, Obst. 70
 —, discussion on pregnancy in a septate uterus, Obst. 192
 — on spontaneous transverse rupture of uterus at fundus during labour, Obst. 138
 —, tubal mole, with œdema of connective tissue of the villi, Obst. 235; report of Pathology Committee on (p. 237); discussion (p. 237): Dr. Lockyer, Mr. Alban Doran
 MAY, W. PAGE.—Discussion on cases of chronic syphilitic poliomyelitis and chronic meningo-myelitis, Neur. 26
 — and Holmes (Gordon M.), exact origin of pyramidal tracts, quoted, Path. 72
 Mayo, W. J., appendix as cause of gastric symptoms, quoted, Surg. 187, 208
 —, causes of appendicitis, quoted, Surg. 181
 —, gastrojejunostomy in cases of mistaken diagnosis, quoted, Surg. 203
 —, spasm of pylorus, quoted, Surg. 205
 MEACHEN, G. NORMAN.—Case of epidermolysis bullosa, Derm. 74; discussion (p. 74): Dr. T. C. Fox
 —, case of urticaria pigmentosa, Derm. 108
 Measles, age-incidence, Epid. 55
 —, case-incidence in nine epidemics of, at a public school, with notes on pre-eruptive symptoms (H. G. Armstrong), Epid. 45
 —, immunity, statistics, Epid. 50
 —, incidence among telegraph messengers, Epid. 49
 —, incubation period, Epid. 49
 —, infective pneumonia in, Epid. 56
 —, Koplik's spots in, Epid. 54
 —, lymphatic system in, Epid. 53
 —, Meunier's sign, Epid. 50
 —, mortality from, Epid. 55
 —, odour in, Epid. 55
 —, prodromal rashes in, Epid. 53
 —, symptoms, Epid. 50
 —, statistics, Epid. 46, 47, 48, 50, 52, 54, 56
 Meat extracts, avoidance in chronic nephritis, Therap. 143
 Meatus (auditory), cartilaginous, pedunculated papilliform growth springing from the posterior border of (W. Milligan), Otol. 44
 —, exostoses and hyperostoses, local distribution, Otol. 77
 —, external, extension through, of malignant growth of right temporal bone (P. W. Williams), Otol. 85
 —, right external, exostosis of, in boy aged 10 (H. Todd), Otol. 76
 —, treatment discussed, Otol. 76, 77
 Meckel's diverticulum containing calculi and producing colic (J. Sherren), Clin. 11
 —, —, —, mistaken for appendicitis, Clin. 12, 15
 —, intestinal concretion found in (A. Carless), Clin. 30
 Mediastinitis fibrosa, Med. 60
 Mediastinum, diseases of, diagnosis, by spinal percussion, Med. 215
 —, growths in or cysts of, diagnosis from pericardial effusion, Med. 63, 65
 —, posterior, new field for diagnosis opened up by dorsal percussion, Med. 238
 —, recurrence of malignant growth in, in treatment by radium (N. S. Finzi and W. Hill), Clin. 159
 Medical assistance, public, compliance with sanitary requirements on part of recipients, advocated, Epid. 35
 Medical practitioners, knowledge of vaccine therapy needed by, R.S.M. 2

- Medical terms, literal as contrasted with specified meanings, *Odont.* 160
- Medicine, a conjectural art if dependent on aid of senses only, *R.S.M.* 75, 76
- , history of, phases in, *R.S.M.* 76
- (recent), relations to bacteriology, *R.S.M.* 3
- (clinical), diagnosis by methods of, not always tenable in cases of bacterial infection, *R.S.M.* 4, 5
- , ignorance of, among bacteriologists, *R.S.M.* 113
- , observations in, as guide to vaccine therapy, *R.S.M.* 124
- Medulla oblongata, chromatolysis of cells of, from case of electric shock, *Path.* 145
- Melena, hypersecretion cause of, *Surg.* 182, 185
- in appendicular gastralgia, *Surg.* 195, 197, 207
- in duodenal ulcer, *Surg.* 75-77, 80, 93, 100, 108
- Melancholia, association with pyorrhoea alveolaris, *R.S.M.* 175
- Melanosis cutis, section of (G. Pernet), *Derm.* 32
- Meltzer and Auer, experiments on monkeys to test value of magnesium-sulphate treatment of tetanus, *Med.* 41
- Membrane, adventitious, resembling normal drum, formed after radical mastoid operation (H. J. Davis), *Otol.* 58
- Membranes and placenta of quadruplets (J. A. Willett), *Obst.* 80
- Men more resistant to cosmic influences than women, *Baln.* 2
- Mena House, as a health resort, *Baln.* 123
- Menard, on metabolic osteo-arthritis, quoted, *Baln.* 8
- Ménière's symptoms, well-defined and transitory, case with; ? migraine with auditory and vestibular phenomena (D. McKenzie), *Otol.* 50
- Meningitis (acute) during secondary syphilis, *Neur.* 37
- (basic, posterior) or ependymitis, symptoms of, followed by internal hydrocephalus and amaurosis without definite ophthalmoscopic changes (F. Parkes Weber), *Child.* 62
- , cause of olfactory discharges, *Neur.* 4
- (cerebrospinal), treatment by anti-serum, *R.S.M.* 85
- , diagnosis from myelitis, *Neur.* 22
- (otitic), *Otol.* 25
- , result of histological examination of petrous bone in (S. R. Scott), *Otol.* 24
- (spinal, syphilitic), *Neur.* 60
- (syphilitic), early appearance, *Med.* 36, 37
- , chronic, with compression of cord (T. Grainger Stewart), *Neur.* 33
- , localized, due to re-infection, *Neur.* 36, 37
- , relation of syphilitic eruption of skin to, *Neur.* 36
- , results of operation, *Neur.* 21
- with compression paraplegia (T. Grainger Stewart), *Neur.* 20
- with universal arteritis, *Neur.* 51
- (and parasymphilitic) lymphocytosis of cerebrospinal fluid in, *Neur.* 39
- (tuberculous), normal or high indices in, *R.S.M.* 192
- Meningo-myelitis, chronic, with internal ophthalmoplegia (W. Harris), *Neur.* 24
- , syphilitic, *Neur.* 60
- Menopause, condition of patients suffering from uterine fibromyomata, prior to and after, *Obst.* 218
- , connexion with adiposis dolorosa, *Clin.* 59, 61
- , occurrence of metabolic osteo-arthritis at, *Baln.* 8
- , relation of shrinking of fibromyoma of uterus to, *Obst.* 214-219
- Menorrhagia, persistent, caused by fibrosis of uterus (J. Inglis Parsons), *Obst.* 240
- , treatment by electricity, *Electr.* 7, 9, 10, 12, 17, 18, 19
- Menstruation, alterations in, in cases of fibromyoma of uterus, *Obst.* 213
- (scanty), treatment by electricity, *Obst.* 20
- Mental defect, spastic diplegia in case of, *Child.* 70
- , non-cretinous, with goitre, case of (D. Forsyth), *Child.* 95

- Mental development, premature, in children the subjects of tumour of pineal body, Neur. 74, 75
- Merchandise Marks Act, Therap. 88
- — — — —, suggestion to make applicable to proprietary medicines, Therap. 100
- Mercury in treatment of syphilis, Surg. 211, 222
- — — — —, antiquity of method, Surg. 211
- — — — —, effect on serum reaction, Surg. 221, 229, 230
- — — — —, treatment by, in tabes and general paralysis, Neur. 42
- — — — —, preparations of, in treatment of syphilis by fumigations, rectal administration, and absorption, Surg. 214
- — — — — by intramuscular injections, soluble, Surg. 212
- — — — — — — — — — —, insoluble, Surg. 214, 233, 235, 245
- — — — — — — — — — —, oral administration (pills), Surg. 230, 240, 243, 244, 247
- — — — — — — — — — —, reasons against, Surg. 230, 234, 235
- — — — — — — — — — — by inunction, Surg. 215, 223, 233, 235
- — — — —, inunction of, in treatment of purpura, Child. 163
- — — — —, atoxylate of, in treatment of syphilis, Surg. 217
- — — — —, cyanide of, intravenous injections in treatment of syphilis, Surg. 248
- — — — —, perchloride of, intravenous injections in treatment of syphilitic poliomyelitis, Neur. 26
- Merfchejewsky, Dr. W. O., on breadth of pelvis in eunuchs, quoted, Path. 121
- — — — —, on the Skoptzi (Russian castrating sect), Path. 105
- Mesenteric region, new field for diagnosis opened up, by dorsal percussion, Med. 238
- Mesentery, chylous cyst of (large) (S. G. Shattock), Path. 98
- — — — —, associated with enlarged glands in mesentery; death from volvulus (H. Burrows), Child. 164
- — — — —, see also *Pancreatic and visceral or mesenteric dulness*
- Metabolism, see also *Osteo-arthritis, metabolic*
- — — — —, inadequate, gouty arthritis connected with, Baln. 8
- Metaplasia, processes of, Otol. 68, 69
- Metchnikoff, infection of chimpanzees with diarrhoeal faecal discharges, Epid. 96, 97
- — — — —, remote bacterial origin of arterial atheroma, quoted, R.S.M. 13
- — — — —, serum-therapy of syphilis, Surg. 229
- Meteorology of the Chilterns, Baln. 34-35
- Metritis, treatment by electricity, Electr. 7-20, 23
- — — — —, with septic endometritis, in slight puerperal sepsis, Obst. 224, 225
- Metrorrhagia, treatment by electricity, Electr. 7, 16, 22
- Meunier's sign in measles, Epid. 50
- Mice, development of tumours in, after protection against implantation of cancer, Therap. 79
- — — — —, possible agency in spread of typhoid fever, Epid. 210
- — — — —, transplanted tumours in, cure of, contrasted with spontaneous invasion, Therap. 81
- MICHELS, E., and WEBER, F. PARKES.—Disappearance of chronic ascites in alcoholic cirrhosis after repeated paracentesis and laparotomy without omentopexy, Clin. 167
- Microbic origin of malignant tumours presumed, Surg. 10
- Micrococcus catarrhalis*, Odont. 31, 33, 36, 37, 39, 40, 41, 44, 46
- — — — —, indifferent definition of characters, R.S.M. 40
- — — — —, opsonic index of, R.S.M. 121
- — — — —, presence in pyorrhoea alveolaris, R.S.M. 173
- — — — —, vaccine prepared from, uses, R.S.M. 114, 115; Odont. 44
- — — — — *neoformans*, invasion in cancer, R.S.M. 18
- — — — — *paratetrageus*, indifferent definition of characters, R.S.M. 40
- Micro-organisms, action on leucocytes, method of studying, Path. 75
- — — — —, attenuation in virulence, Epid. 76
- Micro-organisms, conveyance along electrical current, R.S.M. 156
- — — — — — — — — — — by flies, Epid. 261
- — — — — (extraneous), pathogenetic infection by, R.S.M. 22, 23

- Micro-organisms (extraneous), pathogenetic infection by, methods of determining progress in extinction, R.S.M. 23-26
- , freeing different parts of the body from, R.S.M. 144
- , names of, substitution for names of diseases, R.S.M. 142
- (saprophytic), surface infections of, R.S.M. 22
- , control of, R.S.M. 22
- , virulence of, how increased, Epid. 76
- Micturition, pain and, Obst. 7, 8
- Middle age, nitrous-oxide gas, followed by ether, routine anaesthetic for, Anæsth. 66
- Middlesex Hospital, statistics as to relative frequency of carcinoma of ovary obtained from registers of, Obst. 97
- MIDELTON, W. J.—Discussion on arthritis deformans, Baln. 97
- , discussion on condition of the mouth in chronic disease, Odont. 106
- , on diagnosis and treatment of intussusception in children, Child. 177
- Midwives, intelligence of, Obst. 222, 225, 233, 234
- Milk, advantages of whole milk feeding of infants, Child. 109
- , boiling of, alleged to be no protection against diarrhoeal infection, Epid. 125, 128
- , citrated, in feeding of wasting infants (F. Langmead), Child. 104
- , dilution of, for feeding infants, Child. 105
- , examination of samples of, for bovine tuberculous mastitis, Epid. 220-225
- , flies in, spread of diarrhoea by, Epid. 129
- , restriction of diet to, in chronic nephritis, undesirable, Therap. 140, 143
- , spread of diarrhoea by means of, Epid. 125
- see also *Bacillus bulgaricus*; *Sour-milk treatment*
- vendors, indiscriminate recommendation of lactic-acid bacillus by, Therap. 51, 52
- MILLER, C. H.—Therapeutic value of lactic-acid bacillus, Therap. 61
- MILLER, R.—Case of juvenile general paralysis, with loss of knee-jerks, Child. 134; discussion (p. 134): Dr. Poynton
- , case of lymphadenoma, starting at twenty-fifth month, Child. 135; discussion (p. 135): Dr. Hutchison, Dr. Poynton, Dr. Miller (reply)
- Miller, W. D., fermentation of foodstuffs and causation of dental caries, quoted, Odont. 5
- MILLIGAN, W.—Demonstration of Hays' pharyngoscope, Otol. 9; discussion (p. 10): Dr. H. J. Davis, Dr. Milligan (reply)
- , demonstration of Kuhn's instruments for per-oral intubation, Otol. 49; discussion (pp. 49-50): Dr. Paterson, Dr. StClair Thomson, Dr. Dundas Grant, Mr. Fagge, Dr. Milligan (reply)
- , discussion on adventitious membrane, resembling normal drum, formed after radical mastoid operation, Otol. 59
- , on case of "singer's node" on left cord, Laryng. 107-108
- , on complete inspiratory stenosis of the larynx in a man: case for diagnosis, Laryng. 113
- , on deformity of both pinnae, Otol. 71
- , on exostosis of right external meatus, Otol. 77
- , on pathology of cholesteatomata, Otol. 68
- , on post-operative tests in labyrinthine disease, Otol. 6-9
- , on temporo-sphenoidal abscess, Otol. 74
- , on thickening of the cartilage of both auricles (? othæmatoma), Otol. 53
- , large nasopharyngeal growth in a boy aged 12, Otol. 47; discussion (pp. 47-49): Dr. Paterson, Dr. F. Powell, Mr. Yearsley, Dr. StClair Thomson, Dr. Peters, Dr. Dundas Grant, Mr. Fagge, Dr. Milligan (reply)
- , pedunculated papilliform growth springing from the posterior border of the cartilaginous meatus, Otol. 44; discussion (p. 45): Mr. West, Dr. Dundas Grant, Mr. Macleod Yearsley, Dr. Bronner, Mr. C. H. Fagge, Dr. Milligan (reply)
- MILNE, R.—Antiseptic applications in scarlet fever, Epid. 79
- , case of acromegalic gigantism, Clin. 54

- MILNE, R.—Discussion on control of scarlet fever, *Epid.* 89
 —, scarlet fever: its home treatment and prevention, *Epid.* 57; discussion (p. 65): Dr. Goodhart, Dr. T. Thomson, Dr. Parsons, Dr. F. M. Turner, Dr. Biernacki, Dr. Buchan, Dr. M. Richards, Dr. Crookshank, Dr. Thresh, Mr. Armstrong (reply), Dr. Milne (reply)
 —, two cases of achondroplasia, *Clin.* 54
 Mind-blindness, see *Apraxia*
 Mineral waters containing calcium salts, why diuretic, *Baln.* 9
 —, radio-active, *Baln.* 9
 Misokuchi, results of gastrectomy, *Surg.* 141
 Missenden (Great), medical history of, *Baln.* 42
 Molars (six-year-old), extraction of, case showing result (G. Thomson), *Odont.* 156
 —, extraction, result on facial expression, *Odont.* 157
 —, third impacted, causing neuralgia, diagnosis by skiagrams (C. Schelling), *Odont.* 114
 MOLE, H.—Discussion on exostosis of right external meatus, *Otol.* 76
 Mole, congenital hairy (C. A. R. Nitch), *Clin.* 198
 — (tubal), case of (R. D. Maxwell), *Obst.* 235; report of Pathology Committee on, *Obst.* 237
 — — — with oedema of connective tissue of villi (R. D. Maxwell), *Obst.* 235
 — — — with proliferation of Langhans' layer, *Obst.* 236
 — — —, specimen of (E. W. Hey Groves), *Obst.* 134
 Moles, pigmented, in female child aged 2 (E. G. G. Little), *Derm.* 30
 —, refrigeration in treatment of, *Electr.* 44
 Molesworth, pathology of acne, quoted, *Path.* 173, 174
 Molluscum contagiosum, treatment by Röntgen rays derived from static electricity, *Electr.* 81
 — fibrosum, pigmentation in, *Clin.* 197
 Monilithrix in a boy aged 3 years 9 months (S. E. Dore), *Derm.* 85
 Monkeys, experimental production of snake-venom poisoning in, *Neur.* 106
 —, experiments on, to determine value of magnesium-sulphate solution treatment of tetanus, *Med.* 41
 MOORE, F. CRAVEN.—Discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 111
 — and FERGUSON, R. L.—Rôle of fats in the treatment of disorders of the stomach, *Med.* 25; discussion (p. 35): Dr. Mitchell Bruce, Dr. Hertz, Dr. Saundby, Dr. Hutchison, Dr. Moore (reply)
 MOORE, J.—Partial fixation of left vocal cord presumably of twenty-one years' duration in a male aged 57, *Laryng.* 19; discussion (p. 20): Dr. Watson Williams
 Morel-Lavallée, cases illustrating effects of syphilitic virus on nervous system, quoted, *Neur.* 44
 MORGAN, GEORGE.—Discussion on diagnosis and treatment of intussusception in children, *Child.* 175
 Morgan, W. Parry, and Willcox, W. H., opsonic index of pneumococcus, quoted, *R.S.M.* 121
 Morgan's bacillus, isolation of, *Epid.* 105
 MORISON, ALEXANDER.—Discussion on aneurysm of descending thoracic aorta, *Clin.* 128
 —, discussion on pericardial effusion, *Med.* 100
 — — — on treatment of endocarditis, *Therap.* 114
 Morphia in treatment of puerperal eclampsia, *Obst.* 206
 —, administration in post-operative pain, *Obst.* 9
 Morphine, see also *Scopolamine and morphine*
 Morris, Sir Henry, Bart., leukoplakia and leucoplakic vulvitis becoming carcinomatous, quoted, *Obst.* 29
 MORRIS, Sir MALCOLM, K.C.V.O.—Case for diagnosis, *Derm.* 93
 —, case of lupus erythematosus almost exclusively limited to the scalp, *Derm.* 98
 —, dietetic rules for patients suffering from skin diseases, *Baln.* 77
 —, discussion on case of dermatitis herpetiformis, *Derm.* 90
 — — — of rodent ulcer, *Derm.* 5
 — — — on importance of the Wassermann reaction, *Derm.* 93
 — — — on lupus erythematosus in association with erythema multiforme, *Derm.* 95

- MORRIS, Sir MALCOLM, K.C.V.O., and DORE, S. E.—An unusual condition of the lips, Derm. 18; discussion (p. 19): Dr. Fox, Dr. Pringle
- — — curious affection of the finger- and toe-nails, Derm. 6; discussion (p. 7): Dr. Dore, Dr. Colcott Fox, Dr. Whitfield, Dr. Pringle, Dr. G. Pernet
- — —, pityriasis rubra in a man aged 21, Derm. 60; discussion (p. 61): Dr. Galloway
- — —, report on case of extensive acne with scars, Derm. 7
- — —, thickening of the skin of the palms and soles in a woman aged 31, Derm. 6
- — —, and Fox, W.—Case of lupus erythematosus, Derm. 99
- Mortality, incidence of, heaviest among poorest classes, Epid. 11
- in placenta prævia, foetal, Obst. 175, 176
- — —, maternal, Obst. 169-171
- Mortlake Isolation Hospital, treatment of scarlet fever at, Epid. 83, 92
- MORTON, C. J.—Discussion on examination of intestines by X-rays, Electr. 123
- MORTON, E. REGINALD.—Discussion on simple X-ray stereoscope, Electr. 138
- — — on therapeutical use of statical electricity, Electr. 85
- — — on X-ray treatment of lymphadenoma, Electr. 134
- — —, the relative value of various types of high-tension transformers (including coil) used for the production of X-rays, an address introductory to a discussion on the subject, Electr. 149-154; discussion (p. 155): Dr. Barclay, Mr. A. D. Reid, Dr. Bythell, Dr. Fowler, Mr. S. Gilbert Scott, Dr. Pirie, Dr. Morton (reply)
- — —, treatment of nævi by electricity, cautery and refrigeration, Electr. 37; discussion (p. 47): Dr. Sloan, Dr. L. Jones, Dr. Finzi, Dr. H. Manders, Mr. W. D. Butcher, Dr. McCulloch, Dr. G. B. Batten, Dr. Goffe, Dr. Aldridge, Dr. Morton (reply)
- Morton wave current in treatment of lumbago, Electr. 84
- — — — — neuritis and sciatica, Electr. 83, 84
- — —, see also *Static wave current*
- Morvan's type of syringomyelia, see *Syringomyelia (Morvan's type)*
- Mother and child, syphilis in, treatment, Surg. 231, 232
- and daughter, congenital oedema with cardiac defect in (F. J. Poynton), Child. 66
- Mothers among poorest classes, in towns, ignorance of domestic duties, Epid. 5, 6
- — — — —, instruction as to care of infants and children, Epid. 34
- — — (working-class), due performance of duties to children, Epid. 43
- Mott, F. W., F.R.S., Croonian Lecture on degeneration of neurone, quoted, Path. 27, 28
- — —, on varying amount of change in cells of central cortex from case of transverse lesion of lower cervical region of spinal cord, quoted, Path. 70
- — —, the present position of the neurone doctrine in relation to neuro-pathology, Path. 1-28
- — —, syphilis and parasymphylis of the nervous system, Neur. 35
- — — and Halliburton, W. D., experiments to determine extent of interference with regeneration of motor nerves, quoted, Path. 18-20
- — — and SCHUSTER, EDGAR.—Examination of the brain of a man who lived seven hours after receiving a shock of 20,000 volts, Path. 140
- Mottram, on fatty liver, quoted, Child. 83
- MOUILLOT, F. A. DE.—Discussion on saline waters and common salt, Baln. 84
- MOUNTFORD, J.—Discussion on unilateral overgrowth of lower jaw, Odont. 49
- Mount Vernon Hospital for Consumption and Diseases of the Chest, hospital infection of tuberculosis, as exemplified by the records of the resident staff (J. E. Squire), Med. 137-156
- Mouse-cancer, experimental investigations on, Surg. 11
- Mouse-epithelioma, subsequent graft of, inhibited by extract of mouse-skin, Path. 129
- — — — — by subcutaneous injection of mouse-blood, Path. 129
- Mouse-skin, extract of, inhibiting growth of subsequent graft of mouse-epithelioma, Path. 129
- Mouth, administration of vaccines by, see *Vaccines*, administration method, oral,
- — —, bacterial diseases of, R.S.M. 87
- — —, chronic suppurative lesions in, rheumatic conditions due to, treatment by vaccine, R.S.M. 89, 90

- Mouth, condition of, in chronic diseases (R. Ackerley), *Odont.* 94-103
- , hygiene of, importance in children, *Odont.* 7
- , impression of roof and model of upper and lower jaws from young man (A. R. Tweedie), *Laryng.* 141
- , septic and defective, frequency of, *Odont.* 97, 98
- , skin around, pigmentation in case of pernicious anæmia (H. D. Rolleston), *Clin.* 9
- Mouth breathing, *Laryng.* 41, 49
- Mouth-disinfection, treatment additional to vaccine therapy in pyorrhœa alveolaris, R.S.M. 173
- MOYNIHAN, B. G. A.—Analysis of 197 cases of duodenal ulcer operated on by (1900-1908), (H. Collinson), *Surg.* 79
- , the diagnosis and treatment of duodenal ulcer: an address introductory to a discussion on the subject, *Surg.* 69: discussion (p. 87-116): Sir Lauder Brunton, Dr. W. Hale White, Mr. Herbert Waterhouse, Mr. Frederic Eve, Dr. R. Hutchison, Dr. A. F. Hertz, Mr. Herbert Paterson, Dr. N. Raw, Dr. F. Craven Moore, Mr. C. P. Childe, Mr. Moynihan (reply)
- Muco-pus in middle ear of infants dying from various disorders, question and significance of presence of, *Otol.* 26, 27
- Mucus and blood, passage of, in intussusception, *Child.* 168
- Müller, J., carcinoma of breast, quoted, *Path.* 163
- Müller, gastric cancer involving other organs, quoted, *Surg.* 145
- Müller's manœuvre, use of, in determining proportion of head to pelvis, *Obst.* 114, 125, 126, 128
- MUMMERY, J. HOWARD.—Discussion on susceptibility of teeth to dental caries, *Odont.* 81
- MUMMERY, J. P. LOCKHART.—Case of congenital absence of the left femur, *Child.* 101
- , case of strangulation of the small intestine by a band in a child aged 15 months, *Child.* 37
- , case for diagnosis, *Child.* 24; discussion (p. 24): Dr. Spriggs, Dr. Jewesbury, Dr. Higgs, Mr. Mummery (reply)
- , discussion on case of acute inflammation in an appendix invaginated into lumen of cæcum, *Child.* 26
- — — of congenital enlargement of one limb in brother and sister, *Child.* 44
- — — hemidystrophy, *Child.* 23
- — — of cyst of humerus, *Child.* 99
- — — of extracapsular fracture of neck and thigh-bone in a boy aged 6, *Child.* 22
- — — of fracture of humerus at site of innocent cyst, *Clin.* 195
- — — on diagnosis and treatment of intussusception in children, *Child.* 176
- — — on enormous sarcomatous tumour, *Child.* 156
- — — on idiopathic dilatation of colon, *Child.* 182
- — — on (?) myositis fibrosa, *Child.* 148
- — — on separation and displacement forwards of lower epiphysis of femur, *Child.* 58
- — — on specimen of hypertrophic stenosis of pylorus, *Child.* 100
- — — on X-ray photographs from boy, aged 3, the subject of habitual constipation, *Child.* 66
- , (?) hyperplastic tuberculosis of the pelvic colon, *Clin.* 201; discussion (p. 206): Mr. Gordon Watson, Dr. Rolleston, Mr. Carless, Mr. R. P. Rowlands, Mr. Mummery (reply)
- , BRUCE-PORTER, H. E., and WHITE, W. HALE.—Case of pneumococcal colitis with hyperpyrexia; severe hæmorrhage treated by appendicostomy, *Clin.* 48: discussion (p. 51): Dr. Dalton, Mr. Symonds, Dr. Hale White, Mr. Lockhart Mummery (reply)
- MUMMERY, S.—Discussion on absorption of teeth, *Odont.* 18
- , discussion on case of painful attrition, *Odont.* 120
- — — on unilateral overgrowth of lower jaw, *Odont.* 49
- , some experiments on the relative susceptibility of different teeth to dental caries, *Odont.* 71; discussion (p. 80): Mr. W. Hern, Mr. F. J. Bennett, Dr. Sim Wallace, Mr. Howard Mummery, Mr. D. Gabell, Mr. Geo. Thomson, Mr. P. J. Proud, Mr. J. H. Badcock, Mr. S. P. Mummery (reply)

- Mummy, Egyptian, condition of teeth in skull of, *Odont.* 154
- Munyon's kidney cure, *Therap.* 84
- Murchison, C., communicability of typhoid fever to non-fever patients in hospitals denied by, *Epid.* 108
- Murphy and Cannon on pyloric spasm, quoted, *Surg.* 73
- Murphy, J. B., proctoclysis (continuous saline rectal irrigation), alluded to, *Obst.* 9
- MURPHY, Sir S.—Discussion on bovine tuberculous mastitis, *Epid.* 245
- , discussion on communication of diarrhoea from sick to healthy, *Epid.* 122
- — on hospital infection of tuberculosis, *Med.* 158
- Musca, breeding-places of, *Epid.* 143
- Muscle fibre, striped, tumours of, see *Rhabdomyoma*
- Muscle spindles, condition of, in case of secondary malignant growths affecting spinal roots, *Neur.* 129
- Muscles, abdominal, action on circulation, *Laryng.* 48, 51
- , contractions, produced under application of static electricity, *Electr.* 82
- — (synchronous) of certain muscles forming floor of mouth, objective and rhythmical "clicking" noise in left ear associated with (H. Tilley), *Clin.* 93
- , stimulation by static electricity, method, *Electr.* 80
- Muscular exercise electrically produced, *R.S.M.* 157
- Mutton-bone removed from right bronchus by direct method (H. Tilley), *Laryng.* 127
- Myalgia, treatment by static wave current, *Electr.* 106
- Myasthenia gravis in relation to lymphatism, *Anæsth.* 32, 46-48
- — — — —, anatomical changes, *Anæsth.* 47, 48
- — — — —, mode of death, *Anæsth.* 32, 47
- Myatonia (amyotonia congenita) (A. E. Naish), *Neur.* 95
- Mycosis fungoides, case of (P. S. Abraham), *Derm.* 39
- — — — — (J. L. Bunch), *Clin.* 6
- — — — — (J. H. Sequeira), *Derm.* 35
- — — — — (A. Whitfield), *Derm.* 64
- — — — —, accompanied by plasmoma, *Derm.* 40
- — — — —, history of syphilis in, *Derm.* 40
- — — — —, multiple (J. H. Sequeira), *Derm.* 35
- — — — —, presence of *Staphylococcus aureus* in lesions following, *Clin.* 8
- — — — —, two cases presenting quite different aspects of the disease (J. H. Sequeira), *Derm.* 35
- Myelin, degenerated, chimiotropic action on nerve fibres, *Path.* 26
- , degeneration of, after snake-venom poisoning, *Neur.* 112, 113
- Myelitis, diagnosis from meningitis, *Neur.* 22
- Myocarditis, cardiac syncope following, cause of sudden death in purulent pericarditis, *Med.* 66
- in pericardial effusion, *Med.* 57, 60
- and dilatation of heart, *Med.* 56, 57, 69
- Myoclonus, with spasm of the tongue (W. Harris), *Neur.* 104
- Myoma of uterus, course sometimes painless throughout, *Obst.* 5
- Myomatous tumour of uterus simulating sarcomatous growth (H. Macnaughton-Jones), *Obst.* 103
- — — — —, report on specimen (C. Lockyer), *Obst.* 104
- Myomectomy during pregnancy, case of (H. R. Andrews), *Obst.* 164
- Myopathy, distal type, in boy, also in several members of a family (F. E. Batten), *Neur.* 92, 93
- , infantile type, Batten's frog-child (T. H. Openshaw), *Clin.* 39
- , juvenile type (P. W. Saunders), *Neur.* 100
- (?) Myositis fibrosa, case of (J. W. Carr), *Child.* 147
- Myositis ossificans, case of (T. Thompson), with pathological report by H. Turnbull, *Clin.* 151

- Myositis ossificans traumatica (P. Maynard Heath), Clin. 149
 Myotonia atrophica, paralysis of right vocal cord in (H. Clayton Fox), Laryng. 20
 ———, two cases of (H. G. Turney), Clin. 145
 Myxœdema, case of (G. W. Dawson), Derm. 84
 Myxofibroma of an ovary removed under spinal anaesthesia (W. W. H. Tate), Obst. 73
- Nævi, treatment of, by electricity, cautery, and refrigeration (E. R. Morton), Electr. 27
 ———, cautery puncture in, Electr. 39, 46
 ———, electrolysis in, Electr. 38, 46
 ———, high-frequency spark in, Electr. 40
 ———, refrigeration in, Electr. 41
 Nævoid tumours, symmetrical, plantar, in an infant (J. H. Sequeira), Derm. 22
 Nævus, linear, in a boy aged 18 (S. E. Dore), Derm. 86
 ———, sections from case of (H. Davies), Derm. 105
 ———, of unusual type (nævus acneiformis) (H. Davis), Derm. 68
 ———, with combined verrucose and vascular elements (H. G. Adamson), Derm. 103
 ——— (telangiectatic, facial) treatment by spark application in static electricity, Electr. 80
 Nails, see *Onychia sicca syphilitica*
 NASH, A. E.—Amyotonia (myatonia) congenita, Neur. 95
 Naples as winter-station, Baln. 27
 ———, choice of residence, Baln. 27
 ———, climate, Baln. 14
 ———, cloudiness at, Baln. 18
 ———, death-rate, Baln. 25, 26
 ———, evaporation at, Baln. 19
 ———, fog absent in, Baln. 18
 ———, humidity of air, Baln. 17
 ———, hygiology of (J. J. Eyre), Baln.
 ———, mortality, Baln. 26
 ———, general, high, Baln. 26
 ———, infant, low, Baln. 26
 ———, zymotic, low, Baln. 26
 ———, rainfall at, Baln. 18, 19
 ———, sewerage of, Baln. 23
 ———, temperature of air at, Baln. 15
 ———, topography, Baln. 14
 ———, vital statistics, Baln. 25
 ———, water-supply, Baln. 22
 ———, improvements in, Baln. 22, 23
 ———, wind and atmospheric pressure at, Baln. 19, 20
 Nasal cavities, fauces and larynx, tongue, cheek, and lips, affected by pemphigus vegetans (H. J. Davis), Laryng. 78
 NASH, J. T. C.—Discussion on communication of diarrhoea from sick to healthy, Epid. 129
 NASH, W. G.—Primary hyperplastic tuberculosis of the stomach and duodenum, Clin. 40; discussion (p. 43); Dr. Parkes Weber, Dr. Rolleston, Dr. Camac Wilkinson, Mr. Nash (reply)
 Nasmyth's membrane, Odont. 158, 161
 Nasopharynx, adenoid growths in, suggesting malignant diseases, cases of (J. Dundas Grant), Laryng. 72, 73
 ———, cancer of, early symptoms, Otol. 29, 30
 ———, growth in, large (W. Milligan), Otol. 47
 Nathan, metabolic osteo-arthritis, quoted, Baln. 8
 Neck, chylous cyst of, excision (E. O. Thurston), Path. 97
 ———, glandular tumours of, effect of X-rays on, R.S.M. 156

- Neck, lymphatic glands of, tuberculosis, special site of invasion, Child. 31
 —, malignant disease of, treated by radium (W. Hill), Laryng. 25
 — — growth in, treated with radium (N. S. Finzi and W. Hill), Clin. 159
 —, multilocular cystic hygroma of (C. P. Childe), Child. 157
 —, swelling on left side of, accompanied by dyspnoea (N. Patterson), Laryng. 22
 —, veins in, distension of, as sign of pericardial effusion, Med. 87, 88
 —, see also *Glands, cervical, tuberculous*
 Necrobiosis of tumour, and thrombosis of veins in, with pregnancy complicated by fibromyoma (T. G. Stevens), Obst. 54
 Necrosis, in case of frontal sinus disease (W. H. Kelson), Laryng. 28
 Necrotic tissue, areas of, a hindrance in vaccine therapy of tuberculosis, R.S.M. 148
 Needle, used in intramuscular injections for syphilis, Surg. 236
 Neisser, attempt at serum therapy in syphilis, Surg. 229
 Neoplasm of tonsil (G. S. Hett), Laryng. 2
 — or tuberculosis? of left ventricular band (J. Dundas Grant), Laryng. 16
 Nephritis, acute, association with lymphatism, Anæsth. 55
 — — —, comparison with rheumatoid arthritis, Baln. 102
 — — —, treatment by diuretics difficult, Therap. 145
 — — —, complicating puerperal septicæmia, R.S.M. 59
 —, chronic, diet in, articles to be avoided, Therap. 143
 — — — — — to be included, Therap. 143
 — — —, diminution of œdema in, Therap. 147
 — — —, elimination of nitrogen in, Therap. 142
 — — —, prognosis, question of albuminuria in, Therap. 147
 — — —, treatment by diaphoresis, Therap. 146
 — — —, dietetic, Therap. 139-148
 — — — — by diuretics, how far desirable, Therap. 144, 145
 — — — — by hot-air bath, Therap. 147
 — — —, principles criticized (W. Langdon Brown), Therap. 139-146
 — — —, some principles criticized (W. Langdon Brown), Therap. 137-146
 —, see also *Albuminuria, nephritic*
 Nerve, divided, changes in central end of, Path. 22, 23
 — (fifth), anæsthesia of, and hemianæsthesia of same side caused by pontine thrombosis (H. Campbell Thomson), Neur. 79
 — (laryngeal, left superior), injection of alcohol into, for relief of tuberculosis of larynx with extreme odynphagia (J. Dundas Grant), Laryng. 152
 — (left third), paralysis of, with left hemiplegia (J. Taylor), Neur. 18
 —, peripheral portion, changes in, Path. 22
 Nerve-cells, changes in, due to lesions of cranial and peripheral nerves, Path. 15
 —, means for, immunization against syphilitic virus effected by, Med. 47
 —, nature of, Neur. 47, 48
 Nerve-elements, side-chain molecules thrown off by, Neur. 48
 Nerve-fibres, axolysis of, Path. 25
 —, chemiotropic action of degenerated myelin on, Path. 26
 —, examination of, after snake-venom poisoning, Neur. 112
 —, formation of, Path. 3, 4, 5
 —, origin of, Path. 2, 4, 5
 — — — and growth, Path. 9-12
 —, regenerated, Wallerian degeneration of, in cat, Path. 12, 22, 23, 24
 —, regeneration of, Path. 11, 12
 — — —, part played by sheath-cells in, Neur. 24-26
 Nerve-fibrils composing axon, Path. 21, 23
 —, terminal clubs or bulbs of, Path. 23, 24
 Nerve-roots (spinal), secondary growths affecting (E. F. Buzzard), Neur. 124
 — — — — —, microscopical examination necessary to determine presence of, Neur. 125, 127-129; pathological report (C. M. Hinds Howell), Neur. 125

- Nerve-symptoms accompanying case of probable late secondary specific pharyngitis (J. Dundas Grant), Laryng. 154
- Nerve-terminals, examination of, after snake-venom poisoning, Neur. 113
- Nerve-trunks, effects of magnesium-sulphate solution on injection into, Med. 41, 53
- Nerves (cranial and peripheral) changes in nerve-cells due to lesions of, Path. 15
- (sacral), tumour of ovary pressing on, effects, Obst. 7
- (lower cervical and upper dorsal), affection of posterior roots, tabes ? lateral sclerosis (Harry Campbell), Neur. 34
- , motor, regeneration of, experiments to determine interference with, Path. 18-20
- Nervous disease of undetermined nature (G. Wood and S. A. K. Wilson), Neur. 96
- Nervous system, acute degenerative changes in, due to snake-venom poisoning (W. K. Hunter), Neur. 105-115
- , intensity due to time of action rather than amount injected, Neur. 108
- , process of, Neur. 108, 109
- , anatomy of, demonstration by chrome-silver method of Golgi, Path. 3, 4
- , aptitude of syphilitic virus for attacking, Neur. 43
- , illustrative cases, Neur. 43, 44
- , diseases of, treatment by application of violet-flame discharge, Electr. 77
- , parasyphilis of (F. W. Mott), Neur. 35; discussion (p. 60): Dr. Gordon Holmes, Dr. Wilfred Harris, Dr. Grainger Stewart, Dr. Stanley Barnes
- , factors on which dependent, Neur. 47
- , parasyphilitic diseases of, aetiology, Neur. 42
- , stimulation by static electricity, method, Electr. 80
- , syphilis of (F. W. Mott), Neur. 35; (H. Head), Neur. 49; discussion (p. 60): Dr. Gordon Holmes, Dr. Wilfred Harris, Dr. Grainger Stewart, Dr. Stanley Barnes
- , cases of, within six years of infection (H. Head), Neur. 49
- , classification of forms, Neur. 35
- , concomitant manifestations, Neur. 60
- , prodromal symptoms, Neur. 50
- , treatment, Neur. 60, 63
- , Wassermann's reaction negative, Neur. 61
- (central), disease of, alteration of facial sensation in, Neur. 87
- , case of bilateral abductor paralysis from, Laryng. 162
- , examination of, in case of transverse lesion of spinal cord in lower cervical region, cell-changes, Path. 66
- , histological examination of, in case of transverse lesion of spinal cord in lower cervical region (S. V. Sewell and H. H. Turnbull), Path. 61
- , degenerations, ascending, Path. 64
- , descending, Path. 62
- Nettleship (E.) and Ogilvy, pedigree of familial discoid or "Coppock" cataract, Child. 69
- Neubauer's method of estimation of oxaluria, Med. 11
- Neuralgia, due to impacted wisdom-teeth, diagnosed by aid of skiagrams (C. Schelling), Odont. 114
- (facial), due to pathological conditions of pulps of teeth, Odont. 4
- , treatment by application of violet-flame discharge, Electr. 77
- (trigeminal) of left first and second divisions treated by injections of alcohol (W. Harris), Clin. 200
- (severe) in analgesic facial area, following thrombosis of left posterior cerebellar artery (Wilfred Harris), Neur. 81
- , persistence of pain after removal of Gasserian ganglion, Neur. 87
- Neurasthenia, associated with gastric disorders, Surg. 183, 184
- benefited by lactic-acid administration, Therap. 54
- , choice of anæsthetic for, Anæsth. 67
- , importance of examination of teeth, Odont. 95, 96, 98

- Neurasthenia, misleading term in gynaecological diagnosis, Obst. 3
- Neuritis of brachial plexus, case of, further notes (W. P. Herringham), Clin. 28
- , indication for treatment by static wave-current, Electr. 99, 106
- , treatment by Morton wave current, Electr. 83, 84
- (optic) accompanying tumours of pineal body, Neur. 72
- following treatment by arsenical preparation in syphilis, Surg. 244
- (peripheral), multiple, in child (A. E. Garrod), Child. 38
- Neuroblasts, staining with silver nitrate, Path. 10, 11
- Neurofibrils, origin and development of, Path. 5
- Neurone, conductile element of, Path. 22
- , definition of, Path. 1, 2
- , genetic independence of, confirmed, Path. 13
- , theory of, Path. 2
- , trophic and genetic independence of, established, Path. 27
- Neurone doctrine confirmed by reaction of injury to nerve-cell, Path. 17
- regarding present position in relation to neuro-pathology (F. W. Mott), Path. 1-28
- Neurones, connexion or correlation discussed, Path. 27
- , contact of, and receptive substance or synapse, Path. 28
- , continuity in vertebrata discussed, Path. 27, 28
- , decay of groups of, Neur. 46, 48
- , see also *Nerve-cells*
- Neuro-pathology, present position of neurone doctrine in relation to (F. W. Mott), Path. 1-28
- Neurosis, misleading term in gynaecological diagnosis, Obst. 3
- , spasmodic, ? paramyoclonus multiplex, case of (Arthur Hall), Neur. 28
- , see also *Excoriations, neurotic*
- New growths, pericardial effusion associated with, Med. 56
- Newsholme, A., diarrhoea and density of population, quoted, Epid. 104
- , poverty in relation to phthisis, quoted, Epid. 1, 2
- , transmission of diarrhoeal infection by agency of flies, quoted, Epid. 97
- Newspaper-press under control of patent-medicine vendors, Therap. 84
- Newton, Sir Isaac, results of his adoption of indifferent notation in discovery of differential calculus, R.S.M. 122
- Nissl, changes in nerve-cells, quoted, Path. 15
- NITCH, CYRIL A. R.—Congenital hairy mole, Clin. 198; discussion (p. 198): Dr. James Galloway, Mr. Nitch (reply)
- , discussion on enlargement of tubercle, Clin. 192
- Nitric acid, formation of, in brush discharges, bactericidal agency, Electr. 84
- Nitrogen, elimination of, in chronic nephritis, Therap. 142
- , restriction of, in dietetic treatment of chronic nephritis, Therap. 139-146, 147
- Nitrous oxide, addition of oxygen to, when administered to alcoholics, Anæsth. 68
- not taken well by alcoholics, Anæsth. 68
- gas, administration, under what circumstances dangerous, Anæsth. 71
- , followed by ether, routine anæsthetic for youth and middle life, Anæsth. 66
- NIVEN, J.—Discussion on bovine tuberculous mastitis, Epid. 248
- , discussion on communication of diarrhoea from sick to healthy, Epid. 126
- on control of scarlet fever, Epid. 90
- on summer diarrhoea and enteric fever, Epid. 257
- , methods of, in administering Manchester Milk Clauses (1899), Epid. 220
- , poverty and disease: Part I, The measurements, occasions and relations of poverty to fatal disease; Part II, The relation of poverty to disease in Manchester: presidential address at opening of Epidemiological Section, October 22, 1909, Epid. 1-44
- , summer diarrhoea and enteric fever, Epid. 133-216; discussion (pp. 257-282): Dr. Copeman, Lieut.-Col. A. M. Davies, Col. Notter, Dr. Hamer, Dr. Parsons, Dr. Sandilands, Dr. Wheaton, Dr. Buchan, Dr. Darra Mair, Dr. Niven (reply)
- Nodules on endocardium of heart of infant (R. C. Jewesbury), Child. 45

- Nodules (rheumatic), in case of (probably) gonococcal arthritis (H. Morley Fletcher), Clin. 107
 ———, origin of, discussed, Clin. 108
- Nomenclature of diseases, classification of cysts varies in different editions of, Odont. 161
 ——— of drugs, confusion in, Therap. 90
- NOON, LEONARD.—Discussion on vaccine therapy, R.S.M. 161
- von Noorden, dietetic treatment of nephritis, Therap. 143
- NORMAN, R. H., and WEBER, F. P.—Bluish pigmentation of the skin, Clin. 141; discussion (pp. 141-142); Dr. C. F. Martin, Dr. J. Galloway, Dr. T. Thompson, Dr. H. D. Rolleston, Dr. F. P. Weber (reply)
 ———, case of argyria, Derm. 75
- North Africa and Egypt, influence of climate of, on disease (H. E. L. Canney), Balm. 115
- Nose, bridge of, risk of falling in after submucous resection of septum, Laryng. 57, 58
 ———, cystic swelling at root of (P. M. Heath), Child. 71
 ———, epithelioma of vestibules and adjacent areas, recurrence after surgical removal, radium treatment commenced (W. Hill), Laryng. 26
 ———, fibrosarcoma of, removed after temporary ligature of both external carotid arteries and laryngotomy (W. D. Harmer), Clin. 122
 ———, fissure in mid-line of (rare congenital deformity), in infant (G. Wilkinson), Laryng. 83
 ———, ———, developmental cause, Laryng. 84
 ———, hard palate, fauces and epiglottis, lupus of (W. Stuart-Low), Laryng. 86
 ———, lupus of, treatment by tuberculin inoculation, results, R.S.M. 51, 52
 ———, malignant growths of, method of applying radium to, Electr. 60
 ———, perforation of septum, from salt, NaCl (Dan McKenzie), Laryng. 3
 ——— and throat, lupus of, treatment, discussed, Laryng. 87
- Nostril (anterior), atresia of (Norman Patterson), Laryng. 115
 ———, left, complete occlusion by deflected triangular cartilage in child (G. C. Cathcart), Laryng. 56
 ———, ———, ———, ———, question of operation by removal of projecting cartilage, Laryng. 57
 ———, ———, ———, ———, ——— by submucous resection of septum, Laryng. 56-58
 ——— (posterior), cast of upper jaw from a case of congenital occlusion of (D. R. Paterson), Laryng. 114
- Nostrils, extension of cuticle inside (J. Donelan), Laryng. 90
 ——— (left posterior), congenital occlusion, treatment by operation, Laryng. 91, 92
 ———, obstruction of both, case of (A. Wylie), Laryng. 149
 ——— (posterior), congenital occlusion, case of (D. McKenzie), Laryng. 91
- Nostrums, testimonials and advertisements vaunting, Therap. 86
 ———, traffic in, secrecy essential, Therap. 85
- NOTTER, Col. J. L.—Discussion on summer diarrhoea and enteric fever, Epid. 263
- Nottingham, diarrhoea prevalent in, Epid. 111, 115
 ———, pail-closet system prevalent in, Epid. 111, 112, 113
 ———, typhoid fever prevalent in, Epid. 111, 115
- NOURSE, CHICHELE.—Case of endothelioma of the ethmoid, with microscopical report by V. Wyatt Wingrave, Laryng. 62; discussion (p. 65): Mr. Stuart-Low, Dr. Watson Williams, Dr. Pegler, Dr. Scanes Spicer, Dr. Jobson Home, Mr. Herbert Tilley, Dr. Lambert Lack, Dr. St. Clair Thomson, Dr. William Hill, Dr. Dan McKenzie, Mr. Nourse (reply)
 ———, chronic suppuration of the left frontal sinus, with displacement of the left eyeball and diplopia; operation; recovery, Laryng. 110
 ———, discussion on excision of entire tongue, Laryng. 138
 ——— on removal of a rapidly-growing soft fibroma from posterior wall of left maxillary antrum by a modified Rouge's operation, Laryng. 97
 ———, epithelioma of the larynx in a man aged 69, fourteen months after operation, Laryng. 143

- NOURSE, CHICHELE.—Specific ulceration of the lower lip, inside of the cheek, and tongue, Laryng. 68; discussion (p. 69): Dr. Dundas Grant, Mr. Nourse (reply)
- Nurses, infection with diarrhoea, Epid. 130
- by patients suffering from diarrhoea, Epid. 105, 106
- Nursing Mothers' Aid Society established at Blackburn, Odont. 6
- Obesity, complicating delivery, Obst. 114, 122
- , local, in eunuchs, Path. 122
- , reduction in, due to restriction of salt in diet, Baln. 75
- Obstetrics, progress in, criticized, Obst. 223, 231, 234
- , simpler practices in, when possible, advocated, Obst. 126
- Occupation, classes of, in which phthisis death-rate is high in Manchester, Epid. 29, 30
- Odontocoele (extra-capsular), Odont. 158, 159
- , case of (A. Hopewell-Smith), Odont. 136-141
- (sub-capsular), Odont. 158, 159
- , case of (A. Hopewell-Smith), Odont. 132-136
- Odontocoeles and other cysts of the jaws (A. Hopewell-Smith), Odont. 121-142
- , definition of term, Odont. 160, 161
- , removal by dental surgeons, Odont. 158
- and odontomes, Odont. 160
- Odontoid process, partial luxation of (F. S. Eve), Clin. 78
- Odontological Section (Museum), report of hon. curator (J. F. Colyer), Odont. 143
- Odontoma (epithelial), excision of both maxillæ for (A. Carless), Clin. 10
- Odontomes (follicular), Odont. 161
- , ætiology and pathology of, Odont. 124
- , and odontocoeles, Odont. 160
- Odynphagia, extreme, accompanying tuberculosis of larynx, relieved by injection of alcohol into left superior laryngeal nerve (J. Dundas Grant), Laryng. 152
- in tuberculous subject without any obvious disease of larynx (J. D. Grant), Laryng. 147
- Edema (angioneurotic), family history of, associated with scleroderma of extremities (T. D. Savill), Clin. 33
- (congenital), two cases of, with cardiac defect in mother and daughter (F. J. Poynton), Clin. 66
- , with dilatation of intestinal lymphatics (D. Forsyth), Child. 51
- , of leg (M. Smith), Child. 89
- , diminution in chronic nephritis, Therap. 147
- due to excessive ingestion of salt, Baln. 75
- Esophagus, carcinoma of, case (W. Hill), Laryng. 120
- , skiagrams, Laryng. 120
- , foreign body in, extraction or dislodgment, technique of, Laryng. 76, 77
- (H. J. Davis), Laryng. 75, 76
- , dilatation (large globular) of upper third, above malignant structure (W. Hill), Laryng. 165
- growth in, case of, treated with radium (N. S. Finzi and W. Hill), Clin. 159
- , larynx, four rings of trachea, and part of thyroid gland removed during act of suicide (E. A. Peters), Laryng. 107
- malignant disease of, treatment by radium, Electr. 70
- , —, —, —, —, method of application, Electr. 61
- , pharynx, palate, paresis of, case (W. H. Kelson), Laryng. 90
- , stenosis of (R. H. Scanes Spicer), Laryng. 117
- , upper end of, larynx and lower part of pharynx excised for squamous-celled carcinoma (A. Evans), Clin. 44
- Ogilvy and Nettleship, E., pedigree of familial discoid or "Coppock" cataract, Child. 69
- OGLE, CYBIL.—Discussion on pericardial effusion, Med. 85

- OGLE, CYRIL, and ALLINGHAM, H. W.—Operation on pericardium by gastric incision, Med. 91
- Oil treatment in gastric disorders, Med. 28
- in gastric ulcer, Therap. 134
- Old, J. E. S., on drug known as "Kangalugi," quoted, Therap. 67
- Old age, administration of chloroform in, not recommended, Anæsth. 66
- , cause of pauperism, Epid. 7, 8
- in rural districts, Epid. 7, 8
- (male), atony of bladder, greater trouble in, than enlargement of prostate, Electr. 146
- , voidance of residual urine in, important, Electr. 146
- Olfactory discharges from organic disease, Neur. 2
- OLIVER, GEORGE.—A combination of the auscultatory and tactile methods of reading the arterial pressure (systolic and diastolic), Med. 207; discussion (p. 210): Dr. Mitchell Bruce, Dr. Rolleston, Dr. Oliver (reply)
- (?) Olivo-ponto-cerebellar atrophy, case of (G. Holmes), Neur. 89
- Olshausen, Cæsarean section in eclampsia, quoted, Obst. 196, 197
- Omentopexy and peritoneal drainage for chronic ascites connected with hepatic cirrhosis, patient shown a few months after (F. Parkes Weber), Clin. 80
- Omentum, gastro-hepatic, division of, second step in operation for removal of lymphatic are connected with stomach, Surg. 152
- , great, carrier of cancer-cells to pelvic organs, Surg. 148, 150
- , circulation in, Surg. 163, 164
- , freeing and removal, fifth step in operation for removal of lymphatic area connected with stomach, Surg. 152
- , largest lymph-bearing area connected with stomach, Surg. 147
- , removal important in every radical operation for cancer of stomach, Surg. 129 150-152
- Onychia sicca syphilitica, case of (H. G. Adamson), Derm. 43
- , syphilitic, case of (J. M. H. MacLeod), Derm. 59
- Oöphorectomy, double, result of, on growth of uterus in rabbit (S. G. Shattock), Path. 51
- , hypoplasia of accessory sexual organs following, Path. 119
- , influence on growth of pelvis (S. G. Shattock and C. G. Seligmann), Path. 102-126
- , results in ox, Path. 115
- in pig, Path. 113
- in rabbit, Path. 111
- Open-air life of cows, in relation to bovine tuberculosis, Epid. 232
- OPENSHAW, T. H.—Case of myopathy, infantile type (Batten's frog-child), Clin. 39
- , case of rheumatic spondylitis with torticollis and subluxation, Clin. 37; discussion (p. 38): Mr. Pearce Gould, Mr. C. Symonds
- Ophthalmia (gonorrhœal), treatment by vaccine inoculations, R.S.M. 62
- neonatorum, blindness caused by, expense to state, Surg. 225
- Ophthalmoplegia, internal, chronic meningo-myelitis (W. Harris), Neur. 24
- , transient, in cerebral syphilis, Neur. 52
- Ophthalmoscopic signs absent in case of family amaurotic idiocy (F. Parkes Weber), Child. 58
- of internal hydrocephalus and amaurosis following symptoms of posterior basic meningitis (F. Parkes Weber), Child. 62
- Opisthotonos, accompanying tumours of pineal gland, Neur. 72, 73
- Opium habit in the Chilterns, Baln. 36, 37
- Oppenheimer, hæmorrhage in duodenal ulcer, quoted, Surg. 75
- Oppenheimer's lactigen, Therap. 63
- Opsonic index, R.S.M. 86
- , control of dosage of vaccine by, R.S.M. 32, 33, 47, 116, 142
- , correlation of temperature to, R.S.M. 33, 41, 42
- , diagnosis under, must be complete, R.S.M. 47
- , difficulty of use as guide to dosage of pneumococcus vaccine, R.S.M. 165, 166

- Opsonic index, doctrine of, greatest artificial limitation to vaccine therapy, R.S.M. 119, 143
 ———, as guide to dosage in vaccine therapy, failure of, R.S.M. 98, 121
 ——— (high), cases with no improvement under, R.S.M. 191, 192
 ——— (low), cases of improvement under, R.S.M. 191
 ———, measurement, capacity for accuracy in, discussed, R.S.M. 26-29
 ———, functional error in, R.S.M. 29
 ———, to test progress of extinction of infection during inoculation, R.S.M. 23
 ———, ———, when inoculation is discontinued, R.S.M. 23
 ———, prognosis of pulmonary tuberculosis by, valueless, R.S.M. 200-203
 ——— in relation to fatigue, R.S.M. 74
 ———, relation to temperature, R.S.M. 123, 211, 212
 ———, rise and fall, as correlated with improvement and aggravation in condition of patient, R.S.M. 29-31, 190-192
 ———, value of, R.S.M. 160
 ——— in determining infecting organism, R.S.M. 90, 91
 ——— in diagnosis, R.S.M. 45
 ——— (bacterial), R.S.M. 94, 95, 105, 106, 109
 ———, combined with auto-inoculation experiment, R.S.M. 46
 ———, limitations, R.S.M. 119-124
 ———, in treatment, R.S.M. 47, 48
 ——— in vaccine therapy, R.S.M. 44, 45, 95, 96, 190
- Opsonic power of blood, discovery of, R.S.M. 4
 ———, phagocytic activity dependent on, R.S.M. 164
- Oposonins, production of, R.S.M. 162
 ———, response of, to inoculations of living cultures and emulsions sterilized by heat, R.S.M. 162, 164
- Oposonizing action of serum of healthy rabbits, effect of injection of various tuberculins and of tubercle endotoxin (R. J. Hewlett), Path. 165
- Optic atrophy, caused by congenital lues, which ultimately led to dementia paralytica juvenilis (H. Head), Neur. 33
- Ord, W. M., relation between uterine disorders and arthritis, quoted, Baln. 105
- Organic disease, special sense discharges from (Sir W. R. Gowers, Hughlings Jackson lecture), Neur. 1
- Organo-therapy, failure in cancer, Therap. 70, 71
- ORMEROD, J. A.—Discussion on degenerating changes in nervous system, illustrated by snake-venom poisoning, Neur. 116
 ——— on distal type of myopathy, Neur. 94
 ——— on examination of blood-serum of idiots by Wassermann reaction, Neur. 123
 ——— on secondary growths affecting spinal roots, Neur. 129
- Orr, V. B., heart massage in heart failure during anaesthesia, Anaesth. 1; discussion (p. 10): Mr. R. Gill, Dr. D. Buxton, Dr. A. E. Russell, Dr. E. A. Starling, Dr. Orr (reply)
- Orsudan, injection of, followed by blindness, Surg. 217
 ———, in treatment of syphilis, Surg. 217, 246
- Orth, primary tuberculosis of tonsils, Child. 29
- Orthodiagraph, portable, use of, in diagnosis of pericardial effusion, Med. 82
 ———, value of, in demonstrating causation of gastric resonance, Med. 233
- ORTON, H.—Discussion on examination of intestines by X-rays, Electr. 121
 ———, on radium in treatment of malignant growths, Electr. 70
 ———, discussion on X-ray treatment of lymphadenoma, Electr. 134
- OSLER, Professor W., F.R.S.—Catarrhal jaundice, quoted, Med. 171
 ———, dangers of dental caries, quoted, Odont. 6
 ———, discussion on hospital infection of tuberculosis, Med. 159
 ——— on teaching of therapeutics in hospital wards, Therap. 7
 ——— on thoracoplasty in treatment of empyema, Clin. 92
 ——— on treatment of stricture of rectum and other conditions by fibrolysin, Clin. 89

- Osteitis, alveolar, Odont. 29
- Osteo-arthritis, Baln. 86, 89
- , comparison of granular kidney with, Baln. 102
- , diagnosis by Röntgen rays, Baln. 100, 102
- (metabolic), due to thyroid insufficiency, Baln. 8
- , occurrence at menopause, Baln. 8
- , points of distinction from rheumatoid arthritis, Baln. 100, 101
- Osteomatous growths, attempts to produce by grafting of foetal bones (S. G. Shattock, C. G. Seligmann, and L. S. Dudgeon), Path. 127-140
- Osteomyelitis of the right temporal bone, secondary to mastoid disease; removal; recovery (H. Tod), Otol. 57
- (septic), removal of frontal bone for (C. J. Symonds), Laryng. 40
- (tuberculous), treatment by subperitoneal resection of shaft of ulna (C. P. Childe), Child. 158
- Otorrhœa in scarlet fever, Epid. 62
- Ova, human, attacked or spared by virus of syphilis, Neur. 46
- — — presence of spirochæte in, Neur. 46
- * Ovaries, ablation of, in young, effects on adult, Path. 118
- — —, practised in India, Path. 118
- , cystic degeneration of, supposed toxic origin, R.S.M. 13, 14
- , displacement of, in subinvolution of uterus, Obst. 226, 227, 228, 231
- Ovary, abscess of, probably due to infection by the *Amœba coli* (H. Williamson and J. D. Barris), Obst. 152
- , actinomycosis of, case of (E. H. Shaw), Obst. 146
- , cancer of, secondary, engrafted on pre-existing cystadenoma, Obst. 97
- — —, incidence, compared with cancer of other organs, Obst. 97
- , specimen from (L. G. Anderson), Obst. 72
- , inflammation of, treatment by electricity, Electr. 7, 8, 9, 13, 15, 18
- , innocent growths of, proportion of malignant to (M. A. Scharlieb), Obst. 85-95
- , internal secretion of, affects growth of accessory organs and pelvis, Path. 119, 120
- , malignant growths of, proportion to innocent (M. A. Scharlieb), Obst. 85-95
- — —, series of cases, Obst. 86-89
- — — — —, tabulated, Obst. 92-95
- , maturation of, relation of growth of female pelvis to, Path. 110
- — — — —, observations on, in human subject wanting or indecisive, Path. 111
- , myxofibroma of, removed under spinal anaesthesia (W. W. H. Tate), Obst. 73
- , papilloma of, innocent, Obst. 96
- — —, malignant, Obst. 96
- , pelvic hæmatocele originating from, cases of (C. Lockyer), Obst. 106; (A. E. Giles), Obst. 111
- , tumours of, classification, Obst. 91, 96, 97
- — —, difficulty of diagnosis, Obst. 97
- — —, large, solid, adeno-carcinoma? (A. E. Giles), Obst. 51
- — —, necessity for careful microscopic examination, Obst. 90
- — —, pressing on sacral nerves, effect on lower extremity, Obst. 7
- — —, relative malignancy, Obst. 96
- — —, statistics, Obst. 97
- — —, strangulated, in child; operation; recovery (J. M. G. Swainson), Child. 72
- OVERY, H. BECKETT.—Discussion on oxaluria and calcium-oxalate deposit, Med. 22
- Owbridge's lung tonic, Therap. 89
- OWEN, S. A., and POYNTON, F. J.—Statistics of pericardial effusion from University College Hospital, Med. 129
- Ox, results of oöphorectomy in, Path. 115
- Oxalic acid, composition of, Med. 6

- Oxalic acid, poisoning by, effects, Med. 6, 7
 ———, production of, Med. 4
 ———, toxicity of, Med. 6
 Oxaluria (R. Maguire), Med. 1
 ———, accompanied by albuminuria, Med. 22
 ———, cause of, Med. 3, 7, 8, 21
 ———, estimation by Neubauer's method, Med. 10, 11
 ———, treatment of, Med. 9, 10
 Oxycephaly, cases of (R. Hutchison), Child. 125
 Oxygen, addition to nitrous oxide when administered to alcoholics, Anaesth. 68
 Ozone, formation of in brush discharges, bactericidal agency, Electr. 84
 ———, under discharge of electric wind, Electr. 76, 83, 84
- Pachydermatocele, case of (A. Smith and J. McDonagh), Clin. 76
 Paddington Green Children's Hospital, procedure of infant-feeding at, Child. 107
 PAGE, C. M.—Discussion on the treatment of tetanus, Med. 51
 Pail-closet system prevalent in Nottingham, Epid. 111, 112, 113
 Pain, associated with disorders of female genital organs (H. Macnaughton-Jones), Obst. 1
 ——— of coccygodynia, Obst. 8
 ———, constant, clinical indication of diagnosis of aneurysm of descending thoracic aorta, Clin. 125
 ———, deceptive symptom in gastric cancer, Surg. 134
 ———, diagnostic importance of, Obst. 6, 7
 ———, due to colitis associated with adnexal affection, Obst. 8
 ——— in duodenal ulcer, Surg. 70, 71, 87, 88, 93
 ——— ———, onset, Surg. 102
 ———, epigastric, Obst. 8
 ——— in extremity in cases of angioneurosis, Clin. 106
 ———, frequently slight or absent in serious conditions of female genitalia, Obst. 4
 ———, greater in incipient than in later stages of diseases of female generative organs, Obst. 4
 ——— of inflammatory origin felt when at rest, Obst. 6
 ——— and micturition, Obst. 7, 8
 ———, mystery of, Obst. 2
 ——— in pericarditis with effusion, Med. 106
 ———, post-operative, administration of morphia in, Obst. 9
 ———, prognostic value, Obst. 8
 ———, referred, due to pelvic disease, Obst. 7
 ——— in right iliac and inguinal regions, origin of, Obst. 6, 7
 ———, severity out of proportion to clinical signs and pathological changes, Obst. 5
 ——— in slight puerperal sepsis, Obst. 224
 ———, subjective as well as objective, cause must be sought for, Obst. 3
 ———, symptom of appendicular gastralgia, Surg. 194
 ———, toleration of, great, on part of women, Obst. 3
 ——— of uterine sarcoma, Obst. 8
 Palate, bony part of, congenital absence of, in a girl, Laryng. 163
 ——— (hard), lupus of nose, fauces, epiglottis and (W. Stuart-Low), Laryng. 86
 ———, traumatic (post-operative), perforation through, communicating with floor of left nasal fossa and maxillary antrum (L. H. Pegler), Laryng. 146
 ——— and soft, destructive ulceration with exfoliation of large mesial sequestrum, apparently of specific origin, in tuberculous subject (J. Dundas Grant), Laryng. 71
 ——— (left), peculiar erythema of, in man who had been a considerable smoker (J. Dundas Grant), Laryng. 27
 ———, pharynx and oesophagus, paresis of, case (W. H. Kelson), Laryng. 90
 ——— ——— ———, treatment, Laryng. 90, 91

- Palate (soft), anaesthesia of, always present in cases of dermatitis artefacta in hysterical subjects, Derm. 29
- , (?) leukoplakia of (A. Wylie), Laryng. 82
- , malformation of, case (J. Dundas Grant), Laryng. 131
- , and pharynx, chronic oedematous inflammation of, case of treatment by vaccine inoculation, apparent failure, R.S.M. 36
- , symmetrical papillomata of, with papilloma of larynx (A. Wylie), Laryng. 95
- , ulceration of, case (L. A. Laurence), Laryng. 164
- Palms and soles, thickening of skin of (Sir M. Morris and S. E. Dore), Derm. 6
- Pancreas, cysts of, pancreatic reaction in subjects of, Med. 177
- , cyst of, traumatic, cured by operation and followed twelve years later by medullary leukemia (A. E. Barker), Clin. 121
- , diseases of, diagnostic value of analysis of faeces in (P. J. Cammidge), Med. 185
- , extirpation of, in dogs, Path. 85, 87, 88
- , functional activity, test for, Med. 200, 201
- , insufficiency, tests for, Med. 200, 201
- , invasion by malignant growths from neighbouring organs, Med. 175
- , and liver, cirrhosis of, in case of bronzed diabetes (H. S. French), Path. 94
- , —, and skin, pigmentary deposits in, in case of bronzed diabetes (H. S. French), Path. 94
- , malignant disease of, pancreatic reaction in cases of, Med. 178
- , metastatic deposits of growth from malignant disease in, Med. 175
- , slowly induced changes in, due to intestinal catarrh resulting in diabetes, Med. 171
- , see also *Calculi, pancreatic*
- Pancreatic dullness, Med. 229
- , diagnostic applications, Med. 229
- , as described by Piorry, Med. 217
- (Cammidge) reaction in urine, cause proved experimentally (P. J. Cammidge and H. C. G. Semon), Path. 79-92
- , —, —, experiences of various observers as to, Med. 181, 182, 203, 204, 205
- , —, —, mistakes in method of performance, Med. 204, 205
- , —, —, origin, proved experimentally (P. J. Cammidge and H. C. G. Semon), Path. 79-92
- , —, —, pentosazone crystals in, Path. 80
- , —, —, positive, absent from urine of healthy individuals, Med. 180
- , —, —, results in 1,475 cases (P. J. Cammidge), Med. 163-184
- , —, —, and visceral or mesenteric dullness, Med. 229
- Pancreatitis (acute and subacute), production in dogs by injection of turpentine, Path. 80, 81, 89
- , —, pancreatic reaction in cases of, Med. 164
- , —, chronic and catarrhal jaundice, Med. 171
- , —, and cirrhosis of liver, Med. 174
- , —, causes of, Med. 167, 170
- , —, diagnosis, Med. 166
- , —, chemical pathology of, Med. 167-169
- , —, complicated by malignant disease, Med. 169
- , —, following typhoid fever, Med. 173
- , —, in arterio-sclerosis, Med. 174, 175
- , —, in gastric and duodenal ulcer, Med. 172
- , —, in pernicious anæmia, Med. 172
- , —, in sprue, Med. 172
- , —, interference with fat digestion and absorption, Med. 190
- , —, in tuberculosis, Med. 173
- , —, pancreatic reaction in cases of, Med. 166
- , —, production in dogs by introduction of intestinal bacteria from duodenum into pancreatic ducts, Path. 83, 90

- Pancreatitis (chronic), rarely primary disease, Med. 166
 ———, secondary to disease of intestine, Med. 169
 ———, pancreatic (Cambridge) reaction in urine proved experimentally to be due to, Path. 82, 86, 89, 90
 Pansinusitis (unilateral), case shown six weeks after operation, when all four cavities were operated on at one sitting (StC. Thomson), Laryng. 114
 Papilliform growth, pedunculated, springing from posterior border of cartilaginous meatus (W. Milligan), Otol. 44
 Papilloma of larynx in child of four years' duration cured by tracheotomy and operations by direct laryngoscopy (StC. Thomson), Laryng. 11
 ——— with symmetrical papillomata of palate (A. Wylie), Laryng. 95
 ——— (large) of larynx, case for diagnosis (A. Thorne), Laryng. 92
 ——— of ovary, innocent, Obst. 96
 ———, malignant, Obst. 96
 ——— on the posterior superior quadrant of the right drum (H. J. Davis), Otol. 60
 Papillomata of larynx from boy aged 6 (H. Tilley), Laryng. 124
 ——— of uvula (F. Coleman), Odont. 20
 Paracentesis for relief of purulent pericardial effusion, Med. 85
 ——— in treatment of pericardial effusion, method, Med. 63
 ——— ———, sites selected, Med. 64, 65
 ——— of serous pericardial effusion, Med. 63, 71
 Parakeratosis variegata, case for diagnosis (Sir M. Morris), Derm. 93
 Paralysis (abductor, bilateral), from central nerve disease, case of (D. McKenzie), Laryng. 162
 ——— (left) in man aged 64, case for diagnosis (H. J. Davis), Laryng. 136
 ———, Brown-Séquard's manifestation of spinal syphilis, Neur. 56, 62
 ——— from acute anterior poliomyelitis in children, treatment by faradaic current, Electr. 105
 ——— (general), aetiology identical with that of tabes dorsalis, Neur. 42
 ———, diagnosis by Wassermann's reaction, Neur. 40-42, 61
 ——— from pseudo-general paralysis, Neur. 39
 ———, exaltation and excitement in, possible explanation of, Neur. 48
 ———, increase of proteid in cerebrospinal fluid in, Neur. 39
 ———, infection of two or more individuals with, from same syphilitic source, Neur. 43, 44
 ———, lymphocytosis of cerebrospinal fluid in, Neur. 40, 41
 ———, physical sign common to tabes dorsalis and, Neur. 43, 47
 ———, small percentage of syphilitic individuals attacked with, Neur. 43
 ———, subjects of, inoculated, to test immunity to syphilis, Neur. 48
 ———, succumb easily to terminal invasions by influenza, dysentery and pneumonia, Neur. 49
 ———, transient intellectual activity in, Neur. 48
 ———, treatment with mercury, Neur. 42
 ———, uselessness and injuriousness of antisyphilitic remedies in, Neur. 49
 ———, juvenile, Neur. 123
 ———, case with loss of knee-jerks (R. Miller), Child. 134
 ———, history in cases, Neur. 45, 46
 ———, sex-incidence equal, Neur. 43
 ——— (infantile), relieved by static wave current, Electr. 104
 ——— (laryngeal), following partial removal of thyroid gland (S. Hastings), Laryng. 150
 ——— (left facial), following mild catarrh of middle ear, no perforation (H. J. Davis), Otol. 60
 ——— of left third nerve and left hemiplegia (J. Taylor), Neur. 18
 ——— (ocular), accompanying cases of tumour of pineal body, Neur. 73, 74
 ——— (respiratory), following intraspinal injection of magnesium-sulphate solution in treatment of tetanus, Med. 45, 46, 52
 ——— (pseudo-hypertrophic muscular), case of (A. Manuel), Child. 72
 ——— ——— (W. Carling), Child. 158
 ——— of vocal cord (right) in myotonia atrophica (H. Clayton Fox), Laryng. 20
 Parametritis in slight puerperal sepsis, Obst. 225

- Parametritis, treatment by electricity, *Electr.* 7-20, 25
- PARAMORE, R.—Discussion of criticism of some principles in treatment of chronic nephritis, *Therap.* 148
- ? Paramyoclonus multiplex, case of spasmodic neurosis (A. Hall), *Neur.* 28
- Paraplegia (compression) syphilitic meningitis, case of (T. Grainger Stewart), *Neur.* 20
- due to syphilitic spinal meningitis, question of operating, *Neur.* 63
- and hemiplegia, following syphilitic infection, *Neur.* 59
- , with girdle sensation, manifestation of cerebral syphilis, *Neur.* 55
- Paraspinal bands, *Med.* 221
- Parasyphilis developing in subjects of congenital syphilis, *Neur.* 46
- , physical sign constant to all forms of, *Neur.* 47
- of nervous system, factors on which dependent, *Neur.* 47
- — — (F. W. Mott), *Neur.* 35; discussion (p. 60): Dr. Gordon Holmes, Dr. Wilfred Harris, Dr. Grainger Stewart, Dr. Stanley Barnes
- — —, investigation of suspected case, *Med.* 38
- Parasyphilitic diseases of nervous system, *ætiology*, *Neur.* 42
- Parenchyma, hepatic, *Path.* 149
- Paresis of palate, pharynx and œsophagus, case of (W. H. Kelson), *Laryng.* 90
- — — — —, treatment, *Laryng.* 90, 91
- (spastic), manifestation of spinal syphilis, *Neur.* 55
- PARKINSON, J. PORTER.—Discussion on case of cirrhosis of liver, *Child.* 42
- , discussion on case of compression of trachea by enlarged thymus, *Child.* 47, 48
- — — — — of congenital malformation of heart, *Child.* 50
- — — — — on pericardial effusion, *Med.* 83
- — — — — on specimen of heart of infant showing nodules on endocardium, *Child.* 45
- , specimen of hypertrophic stenosis of pylorus, *Child.* 99; discussion (p. 100): Dr. E. Cautley, Mr. Lockhart Mummery, Dr. C. R. Box, Dr. Parkinson (reply)
- Parotid region (left), congenital tumour in (? cavernous lymphangioma) (F. Parkes Weber), *Clin.* 144
- PARSONS, J. INGLIS.—Fibrosis of uterus, causing persistent hæmorrhagic menorrhagia, *Obst.* 240; report of Pathology Committee, 241
- and CLARE, T. C.—Prolapsus uteri with severe cardiac disease, operated on under local and spinal anaesthesia, *Obst.* 104
- and GLENDINING, BRYDEN.—Adenomyoma of the Fallopian tube, with tuberculous salpingitis, *Obst.* 238; report of Pathology Committee, 239; discussion (p. 240): Dr. Macnaughton-Jones, Dr. Glendining (reply)
- PARSONS, L. G.—Case of congenital acholuric jaundice, *Child.* 136; discussion (p. 137): Dr. Poynton, Dr. Parkes Weber, Dr. Parsons (reply)
- , case of congenital syphilis, *Child.* 138
- , discussion on measles and its pre-eruptive symptoms, *Epid.* 66
- — — on scarlet fever: home treatment and prevention, *Epid.* 66
- — — on summer diarrhoea and enteric fever, *Epid.* 268
- Party-wall pharyngeal cancer (W. Hill), *Laryng.* 142
- Pasteur, Louis, experiments on attenuation of virulence in micro-organisms, quoted, *Epid.* 76
- PASTEUR, W.—Clinical value of orthodiagraph, quoted, *Med.* 233
- , discussion on pericardial effusion, *Med.* 82
- Patent-medicine vendors' power over newspaper press, *Therap.* 84
- Patent medicines, misnomer, *Therap.* 83
- PATERSON, D. R.—Case of endothelioma of temporal bone, *Otol.* 61
- , cast of upper jaw from a case of congenital occlusion of the posterior naris, *Laryng.* 114; discussion (p. 115): Mr. Rose, Mr. Clayton Fox, Dr. Paterson (reply)
- , discussion on acute pemphigus of larynx, *Laryng.* 23
- — — on case of congenital occlusion of left posterior nares, *Laryng.* 91
- — — of extension of cuticle inside nostrils, *Laryng.* 90
- — — of endothelioma of temporal bone, *Otol.* 83

- PATERSON, D. R.—Discussion on complete inspiratory stenosis of the larynx in a man; case for diagnosis, *Laryng.* 113
 —, discussion on early symptoms in case of epithelioma originating near right Eustachian tube, *Otol.* 29
 — — — on Kuhn's instrument for per-oral intubation, *Otol.* 49
 — — — on large nasopharyngeal growth in boy aged 12, *Otol.* 47
 — — — on objective clicking sound in left ear, *Otol.* 37
 — — — on papillomata of larynx from a boy aged 6, removed by direct method, *Laryng.* 98
 — — — on specimen of the face and mouth of a female infant, showing arrest of development of the right half of the tongue, combined with a cleft of the soft palate and a palato-lingual fold, *Laryng.* 101
 — — — on tracheotomy and repeated operations by direct laryngoscopy for papilloma of larynx in a child, *Laryng.* 12
 —, soft foreign body in the bronchus, *Laryng.* 24
 PATERSON, H. J.—An atypical malignant tumour of the uterus, *Obst.* 99; report of Pathology Committee, p. 99; discussion (p. 100): Dr. Cuthbert Lockyer, Dr. Macnaughton-Jones
 —, apparatus for continuous protoclisis, *Obst.* 81
 —, appendicular gastralgia, or the appendix as a cause of gastric symptoms, *Surg.* 187
 —, bilateral tuberculous pyosalpinx, *Obst.* 80; discussion (p. 81): Dr. Macnaughton-Jones
 —, discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 107
 —, radical operation for cancer of pylorus, *Surg.* 158
 —, gastrojejunostomy and hypersecretion, quoted, *Surg.* 182
 —, gumma of the breast simulating malignant disease, *Clin.* 82
 —, recovery after palliative operation for supposed malignant disease of stomach, *Surg.* 138
 —, results of gastrectomy, quoted, *Surg.* 140
 —, rupture of the uterus treated by suture of the rent *per vaginam* and drainage, *Obst.* 58
 PATTERSON, NORMAN.—Atresia of the anterior naris, *Laryng.* 115; discussion (p. 115): Mr. Clayton Fox, Mr. H. Tilley, Mr. N. Patterson (reply)
 —, swelling on left side of neck, accompanied by dyspnoea, in a male aged 28, *Laryng.* 22
 — discussion (p. 22): Dr. Grant
 Pathologists, advances in knowledge of syphilis due to, *Surg.* 239
 Pathology Committee (Obstetrical and Gynaecological section) reports, adenomyoma of the Fallopian tube, with tuberculous salpingitis, *Obst.* 239
 — — — — —, adenoma malignum cysticum cervicis uteri (J. B. Hellier), *Obst.* 103
 — — — — —, tubal mole, with oedema of connective tissue of villi (R. D. Maxwell), *Obst.* 237
 — — — — —, fibrosis of uterus, causing persistent menorrhagia (J. Inglis Parsons), *Obst.* 241
 — — — — —, Dr. Giles's ovarian tumour (A. E. Giles), *Obst.* 53
 Pathology, endowment for research in, advocated, *Obst.* 91
 Patellæ, both, transverse fracture of, with much separation of fragments, treated by operation (F. S. Eve), *Clin.* 78
 Patent remedies, action of American Medical Association with regard to, *Therap.* 99
 Paul, Howard, amyloid degeneration in adenoma of liver, quoted, *Path.* 159
 Pauper children, boarding out of, *Epid.* 40
 Pauperism, causes of, *Epid.* 7-10
 —, estimation of poverty by, not always possible, *Epid.* 8
 Pawlow, J. L., secretory activity in gastric disorders, quoted, *Med.* 26, 30
 PAYNE, J. LEWIN.—Discussion on condition of the mouth in chronic disease, *Odont.* 110
 —, discussion on spindles of von Ebner, *Odont.* 150
 — and EYRE, J. W.—Observations on the bacteriology of pyorrhœa alveolaris and the treatment of the disease by bacterial vaccines, *Odont.* 29
 PEARSON, J. SIDNEY.—Statistics of pericardial effusion from East London Hospital, Shadwell, *Med.* 124

- Pectoralis major, absence of part of (E. I. Spriggs), Child. 102
- Pedigrees, discoid, familial, or "Coppock" cataract, Child. 69
- , telangiectases with epistaxis, Clin. 110
- PEGLER, L. H.—Case of extreme deflection of triangular cartilage, with crest and adhesions, and anterior prickle-shaped spur, treated by septal fissure, Laryng. 128; discussion (p. 129): Dr. C. Potter, Mr. C. Fox, Dr. S. Spicer, Mr. Stuart-Low, Dr. J. Dundas Grant, Dr. L. H. Pegler (reply)
- , discussion on bands between Eustachian tube and pharynx, Laryng. 70
- on case of asthma, improvement after nasal treatment, Laryng. 116
- on case of benign laryngeal growth, Laryng. 94
- of "bleeding polypus" of the inferior turbinate, Laryng. 106
- of extension of cuticle inside nostrils, Laryng. 90
- on endothelioma of ethmoid, Laryng. 66
- on laryngeal vertigo, Laryng. 80
- on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 57
- on rare congenital deformity of nose, Laryng. 85
- on tumour of post-nasal space, Laryng. 161
- , traumatic (post-operative) perforation through the hard palate, communicating with the floor of left nasal fossa and maxillary antrum, Laryng. 146; discussion (p. 146): Dr. Bronner, Mr. Barwell
- Pelikan, on the Skoptzi (Russian castrating sect), Path. 103, 105
- Pelvic distress in women, treatment by electricity, Electr. 7, 9-16, 19, 29
- inflammations, treatment by electricity, Electr. 7, 8-15, 18-20, 25
- organs, great omentum carrier of cancer cells to, Surg. 148-150
- region, disease in pain in other regions due to, Obst. 7
- Pelvimeter, Skutch's, Obst. 113, 121
- Pelvimetry, best method of, Obst. 128
- , knowledge of, important, Obst. 113
- Pelvis, careful examination during pregnancy necessary, Obst. 127
- , contractions, degrees of, classification, Obst. 114
- , first and second degree, methods of delivery generally normal, Obst. 114
- , third degree, method of delivery, operative, Obst. 114
- , fourth degree, method of delivery, operative, extreme measures necessary, Obst. 115
- , of, modern methods of delivery in (E. Hastings Tweedy), Obst. 113
- (female), growth of, relation to maturation of ovary, Path. 110
- , observations on, in human subject wanting or indecisive, Path. 111
- , influence of oöphorectomy on (S. G. Shattock and C. G. Seligmann), Path. 102-126
- in male Belgian hare, effect of castration on, Path. 104, 105
- (male and female), differentiation between, in fœtus, Path. 110
- at puberty, Path. 116
- of eunuchs, breadth excessive, Path. 121
- , Chinese example, Path. 103, 124-126
- , measurements of, Path. 120-124
- , relative proportion of head to, Obst. 114, 126, 127
- Pemphigus, case of (C. C. Claremont), Child. 162
- , acute, of larynx (H. J. Davis), Laryng. 22
- vegetans affecting larynx and fauces, nasal cavities, tongue, cheek, and lips (H. J. Davis), Laryng. 78
- , probable, case of (P. S. Abraham), Derm. 23
- Pencils, infection of scarlet fever by, in schools, Epid. 89
- Penny removed from gullet of young girl (H. Tilley), Laryng. 135
- Pentosazone crystals in pancreatic (Cambridge) reaction in urine, Path. 80
- Penzoldt, fats in treatment of gastric disorders, Med. 25

- Percussion (anterior) for gastric encroachment, Med. 236
 —, columnar or rachidean, Med. 221
 — in diagnosis of pericardial effusion, Med. 62
 — of tuberculosis, Therap. 25
 — (dorsal) for gastric encroachment, Med. 237
 —, map of (1899), Med. 217
 —, Grocco's, Med. 215, 217
 —, Piorry's, Med. 214, 215, 216
 —, new fields for diagnosis opened up by, Med. 238
 —, of stomach, Med. 231
 — (W. Ewart), Med. 211
 —, of thorax (W. Ewart), Med. 211
 —, pelvic, Med. 230
 —, diagnostic applications, Med. 230
 —, visceral (non-pleximetric or by ordinary thoracic conduction), Med. 224
 —, diagnostic applications, Med. 225
 — (spinal), in diagnosis of diseases of mediastinum, Med. 215
 —, surgical aspect, Med. 238
 — (vertebral or spinous process), Med. 222, 223
 —, Med. 220, 221
 —, in diagnosis and surgical diagnosis, Med. 215
 —, surgical aspect, Med. 238
 Percussion note of normal gastric resonance, Med. 233
 — notes, individual of normal vertebræ, Med. 223
 —, diagnostic application, Med. 224
 — signs of amount of pericardial effusion, Med. 100
 — values of whole series of spinous processes, Med. 214
 Perforation, traumatic (post-operative) through hard palate, communicating with floor of left nasal fossa and maxillary antrum (L. H. Pegler), Laryng. 146
 Pericardial dullness in pericarditis with effusion, Med. 107
 Pericardial effusion, alteration of character of cardiogram by intervention of, Med. 101
 — associated with granular kidney, Med. 56, 97
 — with new growth, Med. 56
 — with scurvy, Med. 56
 — with tubercle, Med. 56, 65
 —, blood-stained, Med. 56
 —, significance of, Med. 98
 — in children, Med. 94
 —, diagnosis, Med. 60, 62, 73
 — by aid of X-rays, Med. 63
 — by use of orthodiagraph, Med. 82
 — from adherent pericardium, Med. 102
 — from dilatation of heart, Med. 62, 100, 101, 102
 — from mediastinal growths or cysts, Med. 63, 64
 —, percussion in, Med. 62
 — rests entirely on physical signs, Med. 62
 —, discussion on, Med. 55-103; opened by (p. 55) Dr. S. West, continued by (p. 69) Dr. D. B. Lees, Dr. W. Ewart, Sir John Broadbent, Dr. W. P. Herringham, Dr. W. Pasteur, Dr. J. Porter Parkinson, Dr. Cyril Ogle, Dr. Ironside Bruce, Mr. Lyster, Dr. F. J. Poynton, Dr. Box, Dr. A. Morison, Dr. West (reply)
 —, heart sounds in, Med. 60
 —, myocarditis in, Med. 60
 —, nature of, clinical indications, Med. 97
 —, percussion signs of, amount of, Med. 100
 —, physical signs of, Med. 56-61, 86-88, 98

- Pericardial effusion, physical signs of, influenced by posture, Med. 57, 74
 ———, pulsus paradoxus rare in, Med. 60, 61
 ——— (purulent), associated with empyema, Med. 126-128
 ——— ——— association with pneumonia without empyema, Med. 124
 ——— ——— with pyæmia, Med. 125
 ——— ——— with tuberculosis, Med. 125
 ——— ——— with traumatism, Med. 125
 ——— ———, diagnosis by Röntgen rays, Med. 93, 103
 ——— ———, treatment by paracentesis, Med. 85
 ——— ———, rapid development increases severity of symptoms, Med. 61
 ——— ———, sinking back of heart in, Med. 57, 58, 81
 ——— (serous) absent or rare in rheumatic pericarditis, Med. 77, 79
 ——— ———, chronic, Med. 65
 ——— ———, diagnosis by aid of X-rays, Med. 75
 ——— ———, confirmed by operation, Med. 81
 ——— ———, treatment, Med. 63
 ——— ——— by paracentesis, Med. 63, 71
 ——— ——— ———, method, Med. 63
 ——— ——— ———, sites selected, Med. 64, 65
 ——— ———, inflammatory, Med. 56
 ——— ———, occurring in course of rheumatic fever, Med. 56
 ——— ———, non-inflammatory or dropsical, Med. 56
 ——— ———, statistics from East London Hospital, Shadwell (J. S. Pearson), Med. 124
 ——— ——— from London Hospital (H. L. Tidy, R. S. Woods, and C. E. Zundel), Med. 115
 ——— ——— from Royal Free Hospital (Ivy E. Woodward), Med. 114
 ——— ——— from St. Bartholomew's Hospital (W. Langdon Brown and A. E. Gow), Med. 134
 ——— ——— from St. Thomas's Hospital (C. R. Box and G. C. Butler), Med. 104
 ——— ——— from University College Hospital (F. J. Poynton and S. A. Owen), Med. 129
 ——— ——— from Victoria Hospital for Children, Chelsea (H. F. L. Hugo), Med. 119
 ——— ——— from Westminster Hospital (A. M. Gossage and H. F. Marris), Med. 110
 ——— ———, treatment, operative, Med. 99
 Pericardial sac, distension by effusion, Med. 58
 ——— ———, area of, Med. 59
 Pericarditis, arrest of diaphragmatic movement following, Med. 82, 83
 ———, compression of lower lobe of left lung in, preceding friction and increase of cardiac dullness, Med. 83, 84
 ———, diagnosis of, difficulty, Med. 83
 ———, dilatation of heart without, Med. 95
 ——— ——— (general), result of, Med. 56, 69
 ———, position of, heart-dullness in, Med. 57, 58, 70
 ——— (local), diagnosis of, Med. 100
 ——— (pneumococcal), association with pneumococcal pleurisy, Med. 88, 89
 ——— ———, causes for escape of detection, Med. 89
 ——— ———, characteristics of, Med. 96
 ——— ———, in children, Med. 94, 95
 ——— ———, friction in, how far present, Med. 98
 ——— ——— from abdomen, Med. 81
 ——— ——— in infants, suggested origin, Med. 89
 ——— ———, prognosis, Med. 66
 ——— (primary), with serous effusion, Med. 56
 ———, puncture of dilated heart in, Med. 81
 ——— (purulent), absence of friction-sounds, Med. 72
 ——— ———, accompanying pneumonia, Med. 66
 ——— ———, bacteriology, Med. 67
 ——— ———, becoming quiescent, Med. 66

- Pericarditis (purulent), diagnosis from left-sided empyema, Med. 66
 ———, difficult, Med. 72
 ———, and leucocytosis, Med. 65, 66
 ———, and pyæmia, Med. 65, 66
 ———, following pneumonia, Med. 66
 ———, ———, dangerous, Med. 66
 ———, latent, Med. 79
 ———, ——— and afebrile, Med. 66
 ———, ———, sudden death in, Med. 66
 ———, prognosis, bad, Med. 72
 ———, treatment, Med. 100
 ———, ———, by incision, Med. 67
 ———, ———, choice of site, Med. 68
 ———, ———, difficult, Med. 72
 ———, ———, by paracentesis, Med. 67
 ———, ———, with empyema, Med. 79
 ——— (pyæmic), characteristics of, Med. 97
 ——— (renal) characteristics of, Med. 97, 103
 ——— (rheumatic), cases, Med. 126
 ———, ———, characteristics of, Med. 96
 ———, ———, dilatation of heart in, Med. 56, 57, 69, 77
 ———, ———, effusion absent or rare in, Med. 77
 ———, ———, prognosis, Med. 81
 ———, ———, treatment, local, Med. 71
 ——— (serous), blood examination in, Med. 63
 ———, treatment by drugs, Med. 71
 ———, ———, operative, Med. 99
 ——— (tuberculous), Med. 97
 ———, with effusion, association with other diseases, Med. 105, 116, 124
 ———, ———, bacteriology of, Med. 108, 132, 133
 ———, ———, condition of lungs in, Med. 109
 ———, ———, of pleural sacs in, Med. 108, 112
 ———, ———, course and duration, Med. 105, 110, 118, 129
 ———, ———, cytology, Med. 132, 133
 ———, ———, diagnoses, Med. 131
 ———, ———, pain in, Med. 106
 ———, ———, post-mortem findings, Med. 109, 112, 118
 ———, ———, pulse in, Med. 107
 ———, ———, symptoms and physical signs, Med. 106, 111, 117, 131
 ———, ———, temperature in, Med. 106
 ———, ———, treatment, Med. 109
 ———, ———, operative, Med. 109
 Pericardium, adherent, diagnosis from pericardial effusion, Med. 102
 ———, experimental injections into, results vary with position of body, Med. 57
 ———, operations on, tabulated cases, Med. 90
 ———, ———, epigastric incision, Med. 91, 92
 ———, ———, removal of fourth or fifth left cartilage, Med. 90
 ———, ———, needling, Med. 90
 ———, shape and outline of, revealed by Röntgen rays, Med. 94
 Perichondritis following double mastoid operation resulting in deformity of both pinnae
 (H. J. Davis), Otol. 71
 ——— of thyroid cartilage of unknown origin (H. Barwell), Laryng. 109
 ———, septic, infection of *Bacillus pyocyaneus* in, Otol. 71, 72
 ——— (tuberculous), of cricoid and arytenoid cartilages (H. Barwell), Laryng. 109
 Perimetritis in slight puerperal sepsis, Obst. 224, 227

- Perimetritis, treatment by electricity, *Electr.* 7-20, 25
- Periodontitis, suppurative, chronic, *Odont.* 29
- Periostitis alveolo-dentalis, *Odont.* 29
- Peritoneal drainage and omentopexy for chronic ascites, connected with hepatic cirrhosis; patient shown a few months after (F. Parkes Weber), *Clin.* 80
- growths, specimen of, from case of lymphocythemia, with blood-slides (J. T. Leon), *Child.* 160
- Peritoneum, separation and removal of, in operation for gastric cancer, *Surg.* 129, 150, 151
- (pre-pancreatic), separation of, seventh step in operation for removal of lymphatic area connected with stomach, *Surg.* 153
- , teratoma of, connected with spinal canal (R. Johnson and T. W. P. Lawrence), *Path.* 43
- Peritonitis, cause of death after operations for gastric cancer, *Surg.* 142
- (general), masking presence of tumours in later stages of intussusception, *Child.* 174
- (pelvic), cases illustrating effects of, *Obst.* 229
- (tuberculous), treatment with tuberculin, *R.S.M.* 115
- PERNET, GEORGE.—Discussion on case for diagnosis, *Derm.* 27
- , discussion on case of acroteric sclerodermia, *Derm.* 34
- — — — of blue patches, *Child.* 133
- — — — of congenital oedema of leg, *Child.* 89
- — — — of dermatitis artefacta, *Derm.* 29
- — — — of purpura, *Child.* 163
- — — — of syphilitic dermatitis, *Child.* 160
- — — — on curious case of affection of finger- and toe-nails, *Derm.* 7
- — — — on diagnosis and treatment of intussusception in children, *Child.* 176
- — — — on importance of the Wassermann reaction, *Derm.* 92
- — — — on present position and treatment of syphilis, *Surg.* 234
- , section of melanosis cutis, *Derm.* 32
- Perroncito, changes in central end of cut nerve, *Path.* 23
- Perry, Sir E. C., and Shaw, L. E., hæmorrhage in duodenal ulcer, quoted, *Surg.* 75
- PETERS, E. A.—Bands between Eustachian tube and pharynx, *Laryng.* 69; discussion (p. 69): Dr. Jobson Horne, Dr. Dundas Grant, Dr. Scanes Spicer, Dr. Pegler, Dr. H. J. Davis, Dr. StClair Thomson, Dr. Lambert Lack, Dr. Peters (reply)
- , discussion on large nasopharyngeal growth in boy aged 12, *Otol.* 48
- — — — on vulsellum-catch forceps, *Laryng.* 126
- , epithelioma of the larynx, *Laryng.* 140; discussion (p. 141): Dr. Dundas Grant, Mr. A. Evans, Dr. Peters (reply)
- , larynx, four rings of trachea, and part of thyroid gland and gullet removed during an act of suicide, *Laryng.* 107
- , "singer's node" on left cord, *Laryng.* 107; discussion (pp. 107-109): Mr. Tilley, Dr. Milligan, Dr. F. Powell, Mr. Barwell, Mr. Horsford, Dr. J. Dundas Grant, Dr. StClair Thomson, Dr. Wylie, Dr. Peters (reply)
- PETREN, Professor.—Discussion on case of syringomyelia, with total anaesthesia, *Neur.* 103
- Petrous bones, histological examination in meningitis, results of (S. R. Scott), *Otol.* 24
- — — — preparations of (S. R. Scott), *Otol.* 22
- Phagocytes, activity of, dependent on opsonic power of blood, *R.S.M.* 164
- Phagocytic index, method of estimating, *R.S.M.* 81
- Pharmacy Act, 1868, control over proprietaries containing scheduled poisons, *Therap.* 89
- Pharyngitis, probable late secondary specific, with nerve symptoms, case of (J. Dundas Grant), *Laryng.* 154
- , secondary specific, in young woman (J. Dundas Grant), *Laryng.* 28
- (tuberculous), treatment by ether inhalation without admixture of exhalations from lungs (R. H. Hodgson), *Clin.* 213, 215
- Pharyngoscope (Hays'), *Otol.* 31
- — — — demonstration of (H. J. Davis), *Laryng.* 23
- — — — (W. Milligan), *Otol.* 9

- Pharyngotomy (lateral, Vohsen's method), removal of lymphosarcoma of tonsil by (G. Wilkin-son), Laryng. 86
 ———, with partial laryngectomy for removal of extrinsic cancer of larynx, Laryng. 53, 54
 ———, method, Laryng. 55
- Pharynx and Eustachian tube, bands between (E. A. Peters), Laryng. 69
 ———, lower part of, larynx and upper end of œsophagus, excised for squamous-celled carci-noma (A. Evans), Clin. 44
 ———, malignant growths, method of applying radium to, Electr. 60
 ———, œsophagus and palate, paresis of, case (W. H. Kelson), Laryng. 90
 ——— and soft palate, chronic œdematous inflammation of, case of treatment by vaccine inoculation, apparent failure, R.S.M. 36
 ———, tuberculosis of, case (A. Wylie), Laryng. 58
- Pharmaceutical Society and its relations with the General Medical Council, Therap. 94, 97
- Pharmacopœia Committee, General Medical Council, resolution passed regarding British Pharmaceutical Codex, Therap. 93, 97
- PHILLIPS, LLEWELLYN.—On the treatment of tetanus by the intraspinal injection of a solu-tion of magnesium sulphate, with cases, Med. 39; discussion (p. 51): Dr. Mitchell Bruce, Mr. C. M. Page, Dr. A. E. Russell, Mr. Arthur Barker, Mr. T. P. Beddoes
- Phlebitis, chronic arteritis obliterans of lower extremity associated with (F. Parkes Weber), Clin. 97
 ——— (nodular syphilitic), Derm. 51
 ———, case of (J. E. R. McDonagh), Derm. 72
- Phleboliths, diagnosis of ureteral calculus from, Electr. 94
- Phlebotomy in treatment of puerperal eclampsia, Obst. 205, 207
- Phlegmasia alba dolens, Obst. 223
- Phosphatides (lecithins), group of lipid substances, Neur. 48
- Phosphorus-poisoning, fatty degeneration of liver in, Therap. 66
 ———, jaundice in, Therap. 66
 ———, phosphorus taken as abortifacient, fatal case with unusual subcutaneous hæmorrhages (R. G. Hann and R. A. Veale), Therap. 64
- Phthisis among poorest classes, Epid. 6
 ———, auto-inoculation, experiments in, R.S.M. 25
 ———, beneficial influence of climate of Egypt and North Africa on, Baln. 126
 ———, early gastric disturbances in connexion with simultaneous presence of pyorrhœa alveolaris, R.S.M. 17
 ———, fats in treatment of, Med. 33, 34
 ——— in Manchester, classes of labour in which death-rate is high, Epid. 29, 30
 ———, common lodging-houses, focus of infection for, Epid. 27
 ———, township, death-rates, males, 1901-5, Epid. 22, 23
 ———, females, 1901-5, Epid. 24, 25, 31
 ———, notified cases for, 1907, Epid. 20
 ——— in North Manchester, death-rates, males, 1901-5, Epid. 22, 23
 ———, females, 1901-5, Epid. 24, 25
 ———, notified cases for 1907, Epid. 20
 ——— in South Manchester, death-rates, males, 1901-5, Epid. 22, 23
 ———, females, 1901-5, Epid. 24, 25, 31
 ———, notified cases for 1907, Epid. 20
 ———, infection, increased by aggregation of population, Epid. 28, 29
 ——— mortality in England and Wales, maximum incidence of rate in males and females in urban and rural counties compared, Epid. 23
 ———, poverty factor in causation of, Epid. 1, 2, 6
 ———, public assistance in prevention of, Epid. 42, 43; see also *Tuberculosis, pulmonary*
 ——— (pulmonary), use of term in contradistinction to "pulmonary tuberculosis," R.S.M. 152
 ———, see also *Tuberculosis (pulmonary)*
- Physical Deterioration, Interdepartmental Committee, findings as to results of dental disease, Odont. 5

- Physical development, premature in children the subjects of tumour of pineal body, *Neur.* 74, 75
- Physicians, practice of, syphilis as department of, *Surg.* 242
 — and consulting physicians, relative positions, *R.S.M.* 112
- Piffel, tuberculous infection of tonsils, quoted, *Child.* 29
- Pig, bones and teeth of, effect of madder on, *Odont.* 80, 83
 —, results of oophorectomy in, *Path.* 113
- Pigment, deposits of, in liver, pancreas and skin, in case of bronzed diabetes (H. S. French), *Path.* 94
- Pigmentation (congenital) abnormal, of one eye (N. B. Harman), *Child.* 67
 —, dermic, abnormalities of, characteristic of disorders of internal secretory glands, *Baln.* 5
 —, in Addison's disease due to disturbance of suprarenal glands, *Baln.* 11
 — (bluish) (F. P. Weber and R. H. Norman), *Clin.* 140
 —, influence of sun on, *Baln.* 4
 —, marked development among white individuals adapting themselves to tropical conditions, *Baln.* 5
 —, protects internal secretory glands from excess of stimulation, *Baln.* 5
 —, of face and neck, peculiar form of, in a woman aged 40 (A. Whitfield), *Derm.* 76
 — in molluscum fibrosum, *Clin.* 197
 — of circum-oral skin and of buccal mucosa in pernicious anæmia (H. D. Rolleston), *Clin.* 9
 —, sequel to case, necropsy, *Clin.* 216
 —, see also *Blue patches*
- PIKE, NORMAN, and LAKE, RICHARD.—Summary of the post-operative tests in eight cases of labyrinthine disease, *Otol.* 2; discussion (p. 6): Mr. Baber, Dr. Milligan, Dr. Dan McKenzie; Mr. A. L. Whitehead, Mr. West, Mr. Lake (reply), Dr. Pike (reply)
- Pills, mercury preparation in form of, *Surg.* 230, 234, 235, 240, 243, 244, 247
- Pilocarpin, administration in chronic nephritis, *Therap.* 146
- Pineal body (gland), angio-sarcoma of, case quoted, *Neur.* 70
 —, function obscure, *Neur.* 76
 —, glioma of, case quoted, *Neur.* 70, 71
 —, tumours of (C. M. Hinds Howell), *Neur.* 65
 —, age and sex-incidence, *Neur.* 71
 —, cases described, with clinical histories and post-mortem examinations, *Neur.* 65-69
 —, of, in children, symptom-complex associated with, *Neur.* 74, 75
 —, diagnosis, aids to, *Neur.* 72
 —, malignancy low, *Neur.* 71
 —, mistaken for other conditions, *Med.* 73
 —, mixed forms, *Neur.* 76
 —, nature, *Neur.* 70
 —, removal by operation, *Neur.* 77, 78
 —, symptomatology, *Neur.* 71-75
 —, teratoma of, *Neur.* 76
- Pinnæ (both), deformity of, resulting from perichondritis following double mastoid operation, (H. J. Davis), *Otol.* 71
- Piorry, advances made in dorsal percussion by, *Med.* 212
 —, map of dorsal percussion, *Med.* 214, 215, 216
 —, originator of organic plexigraphy, *Med.* 212
 —, spinal band of, *Med.* 221
- Piorry's nucleus of dullness, *Med.* 217, 226, 234, 235, 236
 —, diagnostic applications, *Med.* 228
- PIRIE, A. H.—Disappearance of enlarged glands in lymphadenoma under treatment by X-rays, *Electr.* 125-131; discussion (p. 132): Dr. S. Sloan, Dr. A. Finzi, Mr. T. Holland, Dr. Barclay, Dr. Reid, Dr. R. Morton, Dr. H. Orton, Mr. McA. Eccles, Mr. Deane Butcher, Dr. McCulloch, Dr. A. H. Pirie (reply)

- PIRIE, A. H.—Discussion on examination of intestines by X-rays, *Electr.* 121
 ——— on relative value of various transformers, *Electr.* 159
 ——— on therapeutical use of static electricity, *Electr.* 85
 ———, new pocket stereoscope for use with X-ray negatives, *Electr.* 139
- PITTS, G. NEWTON.—Discussion on treatment of endocarditis, *Therap.* 115
- Pituitary body (gland), action of, *Therap.* 48
 ———, discovery of value of, to heart's action, *Anæsth.* 2
 ———, extract of, in scleroderma, *Clin.* 33
 ———, tumour of, diagnosed instead of tumour of pineal body, *Neur.* 73
- Pityriasis rubra, reduction of Fehling's solution by urine and alkaptonuria in, *Derm.* 61
- Placenta and membranes of quadruplets (J. A. Willett), *Obst.* 80
 ——— previa, case of, treatment by Cesarean section (J. M. Munro Kerr), *Obst.* 11
 ———, causes of death in, *Obst.* 171
 ———, foetal mortality in, *Obst.* 175, 176
 ———, maternal mortality in, *Obst.* 169-171
 ———, statistics from American Gynecological Society, *Obst.* 169
 ——— from Berlin, *Obst.* 182
 ——— from Rotunda Hospital, *Obst.* 168
 ——— from St. Petersburg, *Obst.* 182
 ———, treatment by Cesarean section, justifiability discussed, *Obst.* 13-15
 ———, limits of (H. Jellet), *Obst.* 167
- Plants, reflex action, without nervous system, *Path.* 29
- Plasma-cell reaction of connective tissue during cancer immunization, *Therap.* 77
- Plasmoma accompanying mycosis fungoides, *Derm.* 40
- Plate and screws in treatment of separation and displacement forwards of lower epiphysis of femur (D. Drew), *Child.* 56
- Pleura, fluid in, presence determined by Grocco's sign, *Med.* 218, 219
 ———, inflammatory diseases of, commonly treated surgically, prognosis in (R. J. Godlee), *Surg.* 49-68
 ———, normal, opening and drainage of, prognosis, *Surg.* 51
 ———, serous effusion into, associated with acute pleurisy, prognosis, *Surg.* 50
- Pleural effusion, nature of, in relation to accompanying pericardial effusion, *Med.* 98
- Pleural sacs, condition in pericarditis, with effusion, *Med.* 108, 112
- Pleurisy (acute), associated with serous effusion into pleura, *Surg.* 50
 ———, adhesions following, pain relieved by static wave current, *Electr.* 104
 ——— in adults, following acute pneumonia, prognosis, *Surg.* 55-61
 ———, pain simulating, due to pelvic disease, *Obst.* 7
 ——— (pneumococcal), association of pneumococcal pericarditis with, *Med.* 88, 89
- Pleuro-pneumonia, epidemic, Ballard's report on, quoted to illustrate characteristic features of communicable diseases, *Epid.* 99
- Plexigraphy (dorsal), *Med.* 212
 ———, early history of, *Med.* 211
 ———, recent history before and since introduction of radioscapy, *Med.* 213
 ———, value of, summarized, *Med.* 238
 ——— (organic), *Med.* 212
- Pleximeter (Sansom's) in delimitation of gastric nucleus of resonance, *Med.* 234
- Pneumococcal infection in infant simulating generalized tuberculosis (J. Walter Carr), *Child.* 14
- Pneumococcus, infection, lesions due to, treatment by vaccine, *R.S.M.* 189
 ———, intestinal invasion by, effect of lactic bacillus on, *Therap.* 60
 ———, opsonic index to, *R.S.M.* 121
 ———, presence in pyorrhœa alveolaris, *R.S.M.* 173
 ———, see also *Empyema, pneumococcal*; *Pericarditis, pneumococcal*
- Pneumococcus vaccine, dosage by opsonic index difficult, *R.S.M.* 165, 166
 ———, failure after use of, *R.S.M.* 189

- Pneumococcus vaccine in treatment of influenza, R.S.M. 70
 ——— of case of painful throat, R.S.M. 184
 ——— of pneumococcal infection of lung, useless, R.S.M. 206
 ——— of pneumonia, R.S.M. 71, 103, 104, 114
 ——— of pyorrhœa alveolaris, Odont. 43
 ——— (autogenous), methods of obtaining, R.S.M. 167
 ———, in treatment of pneumonia, R.S.M. 164, 166, 167-171, 176
 ———, successful where stock vaccine failed, R.S.M. 171
 ——— (stock) in treatment of pneumonia, R.S.M. 164-171, 176
 Pneumonia, accompanied or followed by purulent pericarditis, Med. 66
 — (acute), in adults, followed by acute empyema, prognosis, Surg. 55-61
 —, general paralytics easily succumb to, Neur. 49
 —, infants suffering from, attacked with diarrhœa, Epid. 107
 —, infective, in measles, Epid. 56
 — (lobar), displacement of heart in two cases of (W. P. Herringham), Clin. 25
 — and scarlet fever, Epid. 62
 —, treatment by pneumococcus vaccine, R.S.M. 71, 103, 104, 144
 ——— by vaccines (stock and autogenous), R.S.M. 164, 166, 167
 ———, cases illustrating, R.S.M. 167-171, 176
 — without empyema, purulent pericardial effusion associated with, Med. 124
 Pneumopericardium, case of, Med. 81, 103
 Polar action (chemical) of direct current, Obst. 16, 17
 Poliomyelitis (anterior, acute), involving the four limbs, two cases (A. Manuel), Child. 21
 — (syphilitic, chronic), cases of (W. Harris), Neur. 23
 ———, treatment by intravenous injection of perchloride of mercury, Neur. 26
 Politzer, Professor Adam, letter from, to Otological Section, Otol. 17
 —, receipt from Otological Section of illuminated address on fiftieth anniversary of obtaining degree of Doctor of Medicine, Otol. 17
 Polygraph, in diagnosis of endocarditis, Therap. 101
 Polyneuritis, alcoholic chromatolysis of Betz cells in, Path. 17
 —, methods of treatment promoting recovery from, Path. 18
 Polypus (adenofibromatous) of uterus showing early malignant disease (J. H. Targett), Obst. 155
 — (aural), malignant growth of right temporal bone resembling (P. Watson Williams), Otol. 85
 —, "bleeding" of the inferior turbinate (S. Hastings), Laryng. 102-106
 — (mucous), presenting at pharyngeal orifice of left Eustachian tube in man suffering from bilateral chronic adhesive otitis media (J. Arnold Jones), Otol. 55
 Polyuria, symptom of rupture of tunica vaginalis in hydroceles, Surg. 167
 Poncet, rupture of tunica vaginalis in hydroceles, quoted, Surg. 170, 172
 Poorest classes, children of, condition, Epid. 11, 12
 —, ill-effects of alcohol among, Epid. 6, 7
 —, in towns, liability to disease, Epid. 4, 5
 —, phthisis among, Epid. 6
 —, schemes for amelioration, proper aims of, Epid. 33
 Poor Law Commission, recommendations of minority report, Epid. 43
 —, reports of, Epid. 1
 Population, aggregation of, phthisis infection increased by, Epid. 28, 29
 Posterior signs of pericardial effusion, Med. 99
 Post-graduate demonstrations in union hospitals advocated, Epid. 35
 Post-nasal space, tumours of, in man (H. F. Powell), Laryng. 160
 Post-operative tests, summary of, in eight cases of labyrinthine disease (R. Lake and N. Pike), Otol. 2
 Posture in anæsthetization of alcoholics, Anæsth. 69
 —, influence on physical signs of pericardial effusion, Med. 57, 74

- Potassium, iodide of, in treatment of syphilis, Surg. 230
- Potential, in atmospheric electricity, Baln. 51, 55-58
- Pott, Percivall, rupture of tunica vaginalis in hydroceles, quoted, Surg. 165
- POTTER, COUBRO.—Discussion on deflection of triangular cartilage treated by septal fissure, Laryng. 129
- , and McDONAGH, J. E. R.—Chancere of the tongue, Derm. 51
- Poverty, cause of disease, Epid. 7
- , classes of disease responding most readily to, Epid. 18
- , definition of term, Epid. 2
- , estimation of, by amount of medical aid received not always possible, Epid. 2
- , by house-to-house inquiry, Epid. 3, 12
- , by number of deaths from sickness occurring in union hospitals, Epid. 2, 3
- , by pauperism not always possible, Epid. 3
- , factor in causation of phthisis, Epid. 1, 2, 6
- , (intense), disease cause of, Epid. 7
- , in London, analysis of immediate causes, Epid. 14
- , method of estimation by Mr. C. Booth, Epid. 4, 14, 15
- , in Manchester, method of estimation, Epid. 13
- , proposals for reduction of, Epid. 32
- , public assistance in prevention of, Epid. 42, 43
- , relation to disease in Manchester, Epid. 15
- , in York, method of estimation by Mr. Rowntree, Epid. 4, 12, 13
- POWELL, H. W. FITZGERALD.—Discussion on adventitious membrane, resembling normal drum, formed after radical mastoid operation, Otol. 59
- , discussion on case of audible tinnitus, Otol. 2
- , of extensive tuberculosis of larynx, Laryng. 89
- , of laryngeal paralysis, Otol. 150
- , of "singer's node" on left cord, Laryng. 108
- , of tuberculosis of pharynx, Laryng. 60
- , on chronic glanders in a man, Laryng. 2
- , on complete inspiratory stenosis of the larynx in a man, case for diagnosis, Laryng. 113
- , on extrinsic carcinoma of larynx, Laryng. 54
- , on large nasopharyngeal growth in a boy, Otol. 47
- , on laryngeal vertigo, Laryng. 80
- , on papillomata of larynx from a boy aged 6, removed by direct method, Laryng. 98
- , on probable late secondary specific pharyngitis, Laryng. 154
- , on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 57
- , on specimen of goitre and portions of larynx and trachea from woman aged 37, Laryng. 38
- , on treatment of tuberculous laryngitis by galvano-puncture, Laryng. 122
- , on tuberculosis of larynx in girl, Laryng. 127
- , on two cases of hoarseness, Laryng. 41
- , on vulsellum-catch forceps, Laryng. 125
- , laryngeal growth, probably malignant, in a man aged 61, Laryng. 74; discussion (p. 74): Dr. Dundas Grant
- , tumour of post-nasal space in a man aged 27, Laryng. 160; discussion (p. 161): Dr. Dundas Grant, Dr. Watson Williams, Mr. C. Fox, Dr. Pegler, Dr. Fitzgérald Powell (reply)
- POWELL, LLEWELLYN.—Discussion on choice of anæsthetic, Anæsth. 76
- Power, D'Arcy, experiments as to absorption of albumin, quoted, Therap. 139
- POYNTON, F. J.—Discussion on case of congenital acholuric jaundice, Child. 137
- , discussion on case of early lymphadenoma, Child. 136
- , of juvenile general paralysis, Child. 134

- POYNTON, F. J.—Discussion on case of rheumatoid arthritis, with albuminuria, and with great enlargement of liver, Clin. 62
- on cases of *adiposis dolorosa*, Clin. 60
- on pericardial effusion, Med. 94
- on "rheumatic nodules" in probable gonococcal arthritis, Clin. 108
- on use of citrated milk for wasting infants, Child. 123
- , rheumatic pericarditis, quoted, Med. 69, 70, 75
- , two cases of congenital oedema, with cardiac defect, in mother and daughter, Clin. 66; discussion (p. 67): Dr. Parkes Weber
- and OWEN, S. A.—Statistics of pericardial effusion, from University College Hospital, Med. 129
- and TROTTER, WILFRID.—Case of cardiolysis, Clin. 199
- Præcordial dullness, Med. 58, 59
- , pear-shaped, Med. 59
- , upward extension, Med. 98
- Pregnancy, character of labour inseparable from health and physical state during, Obst. 127
- (ectopic), difficulty of diagnosis from chronic tubal inflammatory disease, Obst. 237
- , examination of pelvis during, care in, necessary, Obst. 127
- of blood-serum during, by Wassermann reaction, Neur. 121
- and fibromyoma, thrombosis of veins and necrobiosis of tumour (T. G. Stevens), Obst. 54
- , functional activity of thyroid gland essential to, Baln. 10
- , myomectomy during, case of (H. R. Andrews), Obst. 164
- (ovarian), specimen of (E. Hastings Tweedy), Obst. 132
- , prolongation beyond normal time, risk to mother and child, Obst. 127
- in septate uterus, treated by abdominal hysterectomy (W. W. H. Tate), Obst. 190
- , syphilis complicating, treatment, Surg. 237, 238
- , termination of, influence on course of puerperal eclampsia, Obst. 202
- (tubal), decidual reaction extending from endometrium to tubal mucosa, cause of, Obst. 238
- , microscopic appearances in (T. Carwardine), Obst. 157
- , see also *Mole, tubal*
- , tubo-abdominal, case of (F. J. McCann), Obst. 149
- , see also *Vomiting of pregnancy*
- PRENTICE, H. R.—Discussion on syringomyelia with total anaesthesia, Neur. 103
- , ? syringomyelia with Brown-Séquard syndrome, Neur. 100
- Prescott, W. H., abstinence from salt among Tlascalans (of South America), quoted, Baln. 60
- Preservatives, food with, avoidance in chronic gastric ulcer, Therap. 132
- PRIDEAUX, W. DE C.—Case of rupture of a gastric ulcer while in a dental chair, Odont. 91
- , case of vertical fracture through the mandible and canine tooth, Odont. 92; discussion (p. 93): Mr. W. Hern, Mr. W. de C. Prideaux (reply)
- Priessnitz, empiric employment of hydrotherapeutic measures by, Baln. 6
- PRINGLE, J. J.—Discussion on case for diagnosis, Derm. 27
- , discussion on case of acroteric scleroderma, Derm. 34
- of dermatitis herpetiformis, Derm. 90
- of lichen planus annularis, Derm. 68
- of lupus disseminatus faciei, Derm. 62
- of multiple keloid associated with "neurotic excoriations" of "dug-out" type of Colcott Fox, Derm. 42
- of mycosis fungoides, Derm. 40
- of (?) mycosis fungoides, Derm. 64
- of onychia sicca syphilitica, Derm. 44
- of xeroderma pigmentosa with unusual features, Derm. 58
- on elephantiasis associated with tertiary syphilis, Derm. 83
- on lupus erythematosus in association with erythema multiforme, Derm. 95
- on non-ulcerative tuberculides following lupus vulgaris, Derm. 52

- PRINGLE, J. J.—Discussion on symmetrical plantar naevoid tumours, *Derm.* 22
 —, discussion on unusual condition of lips, *Derm.* 20
 — on curious case of affection of finger- and toe-nails, *Derm.* 7
 — on two cases for diagnosis, *Derm.* 72
- PRITCHARD, ERIC.—Discussion on X-ray photographs from boy, aged 3, the subject of habitual constipation, *Child.* 66
 — and STEPHENSON, SYDNEY.—Case of Graves' disease in a lad aged 8, *Child.* 73
- PRITCHARD, URBAN.—Discussion on pathological changes found in ear of deaf mutes, *Otol.* 13
 —, discussion on exostosis of right external meatus, *Otol.* 77
 — on pathology of cholesteatomata, *Otol.* 69
 — on twenty specimens of chronic middle-ear suppuration, *Otol.* 43
 — on well-defined Ménière's symptoms, *Otol.* 52
- Privy-middens, relation to summer diarrhoea, *Epid.* 134
 — and house-flies in connexion with prevalence of typhoid fever, *Epid.* 187
- Proctoclysis, continuous saline rectal irrigation, apparatus for (H. J. Paterson), *Obst.* 81
 —, value of, *Obst.* 9
- Profeta, law of, in congenital syphilis, *Neur.* 45
- Prognosis in certain inflammatory diseases of lungs and pleura commonly treated surgically (R. J. Godlee), *Surg.* 49-68
 — of pulmonary tuberculosis by opsonic index valueless, *R.S.M.* 200-203
 —, value of post-operative pain in, *Obst.* 8
- Proprietary medicines (non-secret), *Therap.* 87
 —, patent and secret remedies, discussion on, opened by Professor W. E. Dixon, continued by Mr. Beddoes, Mr. Gamble, Dr. Duncanson, Dr. Martin, Mr. Woolcock, Mr. Harrison, Professor Cushny, Professor Dixon (reply), *Therap.* 82-100
 —, laws controlling, *Therap.* 88
 —, policy suggested with regard to, *Therap.* 89
 —, statistics relating to, *Therap.* 83
 — remedies, classification, *Therap.* 83
 —, poisonous, how controlled, *Therap.* 89
- Prostate, diseases of, passage of urethral electrode not recommended in, *Electr.* 147
 —, (enlarged), atony of bladder greater trouble to male old age than, *Electr.* 146
 —, adenomatous, treatment by static wave current, *Electr.* 102
 —, fibroid and fibromyomatous, treatment by static wave current not effectual
Electr. 102
 —, obstruction of, relief of symptoms by electrical treatment (J. S. Bolton), *Electr.* 141-146
 —, cases described, *Electr.* 143-145
- Prostatitis (acute) following gonorrhoea, treatment by static electricity, *Electr.* 148
- Prostitution, state control of, in prophylaxis of syphilis, reasons for failure, *Surg.* 228
- Proteid, increase in cerebrospinal fluid in general paralysis and tabes, *Neur.* 39
- Proteids in intestinal tract, abnormal putrefaction, *Therap.* 55
 —, cases dependent upon, benefited by lactic-bacillus administration,
Therap. 54, 55
- Protein, amount ingested, amount of urea excreted by kidney depends on, *Therap.* 141
 — diet in chronic nephritis, *Therap.* 140, 147
 —, limitations should not be too rigid, *Therap.* 142
 —, physiological minimum in diet not necessarily the optimum, *Therap.* 141
 — substances, normal aid of cancer-cells, consequences following inoculation, *Therap.* 77
- Proteus vaccine, in treatment of tuberculous ulceration of skin, *R.S.M.* 21
- Proud, P. J.—Discussion on susceptibility of teeth to dental caries, *Odont.* 82
- Pruritus, case of (G. W. Dawson), *Derm.* 16
 — in leucoplakic vulvitis, *Obst.* 31, 39, 48
 — (anal), treatment by vaccine, *R.S.M.* 15
 — (vulvar and anal), treatment by electricity, *Electr.* 7, 11, 13, 15, 16, 27
- Pseudo-diphtheria, present in pruritus ani, *R.S.M.* 15

- Pseudo-general paralysis, diagnosis from general paralysis, Neur. 39
- Pseudo-immunizers, operations of, R.S.M. 79, 141
- Pseudo-tabes, diagnosis from tabes, Neur. 39
- Pseudo-xanthoma elasticum, case of (E. G. Graham Little and J. H. Sequeira), Derm. 69
- Psoriasis, treatment by electro-vaccination, R.S.M. 155
- by static electricity, Electr. 82, 83
- Puberty, differentiation between male and female pelvis becomes marked at, Path. 110
- , rapid growth of eunuchs at time of, Path. 106
- Pubiotomy, Döderlein's method, Obst. 118
- , merits of, Obst. 116
- , method of delivery in third degree of contracted pelvis, Obst. 115
- , subcutaneous, risks of, Obst. 124
- , operation described, Obst. 117
- Public assistance in prevention of poverty and disease, Epid. 42, 43
- Public health, bearing of dentistry upon, Odont. 1-8
- in Great Britain benefited by cycle of prosperity (1871-80), Epid. 10, 11
- Puerperal eclampsia, causes of, Obst. 204
- , clinical differentiation of types of, Obst. 206
- , geographical distribution, effect on treatment, Obst. 206
- , post-partum cases, Obst. 205, 206
- , prognosis in, difficulty of, Obst. 200, 203
- in relation to rapid delivery, Obst. 200
- , statistics from Glasgow Maternity Hospital, Obst. 201
- from London Hospital, Obst. 201
- , time of occurrence in pregnancy, quoted, Obst. 199
- , treatment by Bossi's mechanical dilatation of cervix, Obst. 204, 205
- by Cesarean section (F. J. McCann), Obst. 193-202
- , effects on fits, Obst. 200
- , historical account, Obst. 196, 197
- , successful case of (F. J. McCann), Obst. 194
- , divergence in methods, Obst. 193
- with morphia, Obst. 206
- by phlebotomy, Obst. 205, 207
- by veratrum, Obst. 205, 206
- septicæmia, treatment by vaccine, R.S.M. 59, 63
- Puerperal morbidity, diagnosis of, Obst. 221, 222
- in German obstetric clinics, Obst. 222
- Puerperal sepsis, death-rate from, improvement in, Obst. 232
- , lymphatic, Obst. 223
- (slight), accelerated pulse-rate and slight rise of temperature in, Obst. 223
- , diagnosis, Obst. 225
- , foul-smelling lochial discharge in, Obst. 224
- , pain in, Obst. 224
- , pathological processes of, Obst. 224-227
- , pelvic conditions resulting from (Sir W. J. Sinclair), Obst. 221-231
- , prophylaxis, Obst. 225
- , symptoms, Obst. 223-225
- Pulp (dental), diseased conditions of, producing neuralgia, Odont. 4
- Pulse-rate, accelerated, in slight puerperal sepsis, Obst. 223
- Pulsus paradoxus, Med. 100
- , rarity in pericardial effusion, Med. 60, 61
- Puncta lachrymalia, bilateral absence of (L. Cole-Baker), Child. 159
- Puncture, simple and electric, of the heart, Anæsth. 5
- Purgative, see *Aperient*
- Purins, secretion of, Therap. 143

- Purpura, case of (C. C. Claremont), Child. 163
— fulminans, case of (J. D. Rolleston and T. McCrick), Child. 75
— ———, diagnosis from hæmorrhagic diphtheria and hæmorrhagic small-pox, Child. 77
— (Henoch's), definition required, Child. 176
— ———, diagnosis from intussusception, Child. 168, 173, 174, 176
— ———, treatment by mercury inunction, Child. 163
PUNSHOW, C. E.—Discussion on Cæsarean section in placenta prævia, Obst. 178
— ———, discussion on very large cervix fibroid, Obst. 185
PUS, gravitation to diaphragmatic region dangerous, Obst. 8, 9
Pyæmia and purulent pericarditis, Med. 65, 66
— ———, purulent pericardial effusion associated with, Med. 125
— ———, treatment by vaccine, R.S.M. 100, 101
— ———, see also *Pericarditis* (*pyemic*)
Pyæmic knee- and shoulder-joints treated by tapping and incision (E. M. Corner and M. Huggins), Clin. 120
Pylorus, cancer of, hypersecretion in, Surg. 186
— ———, treatment, by radical operation (E. W. Hey Groves), Surg. 117-158
— ——— ——— in two stages, illustrative cases, Surg. 119-132, 162
— ——— ——— operative by radical operation, remote results, Surg. 155, 156
— ———, see also *Stomach, cancer of*
— ———, spasm of, reflex and protective in duodenal ulcer, Surg. 73
— ———, symptom of appendicitis, Surg. 187, 205, 207
— ———, stenosis of, caused by ulcer, Surg. 185
— ———, hypertrophic association with cyclic or recurrent vomiting, case of (A. E. Russell), Child. 78
— ——— ——— ——— ———, autopsy, Child. 80
— ——— ———, muscular spasm in, Child. 84, 85
— ——— ———, specimen (J. Porter Parkinson), Child. 99
— ——— ———, treatment, medical or surgical, discussed, Child. 100
— ———, ulcer of, symptoms, Surg. 184
Pyorrhœa alveolaris, association of gastric disturbances in early phthisis with simultaneous presence of, R.S.M. 17
— ——— ——— with melancholia, R.S.M. 174, 175
— ——— ———, bacteriology, R.S.M. 173
— ——— ——— (J. W. Eyre and J. L. Payne), Odont. 29
— ——— ———, diseased conditions included under, R.S.M. 86
— ——— ———, infective nature, R.S.M. 174
— ——— ———, treatment by bacterial vaccines (J. W. Eyre and J. L. Payne), Odont. 29; R.S.M. 148, 149, 172, 176
— ——— ——— ——— and mouth disinfection, R.S.M. 173
— ———, results of, Odont. 3
Pyosalpinx, bilateral tuberculous (H. J. Paterson), Obst. 80
Pyrotartaric acid, Med. 6

- Quack medicines, Therap. 83
 Quadruplets born at the twenty-sixth week (C. White), Obst. 79
 —, placenta and membranes of (J. A. Willett), Obst. 80
 Queen Charlotte's Hospital, results of induction of premature labour for contracted pelvis, Obst. 125
 Quinine in treatment of acute endocarditis, Therap. 114
 — of syphilis, Surg. 237
 — (tasteless), quack remedy, Therap. 84

- Rabbit, results of oöphorectomy in, Path. 111
 —, uterus of, result of double oöphorectomy upon growth of (S. G. Shattock), Path. 51
 Rabbits, experiments on, to test opsonic response to inoculations of living cultures and emulsions sterilized by heat, R.S.M. 162
 —, healthy, serum of, effect of injections of various tuberculins, and of tubercle endotoxin, on opsonizing action of (R. T. Hewlett), Path. 165
 —, inoculation with vaccines to test effects, R.S.M. 81
 — (young), grafting of foetal bones into, Path. 131
 —, see also *Dogs*
 Race, influence of climate on, Baln. 2, 3
 Radiographs, film, method of ascertaining relative position of unerupted teeth by (C. A. Clark), Odont. 87
 Radiography, importance of, in determining presence of urinary calculus, Med. 22, 23
 —, in diagnosis of impacted wisdom teeth, causing neuralgia (C. Schelling), Odont. 114
 —, see also *Röntgen rays*
 Radioscopy, recent history of plexigraphy before and since introduction of, Med. 213
 Radio-vaccination, see *Electro-vaccination*
 Radium, and carbon-dioxide crayon, comparison of results in treatment of naevi, Electr. 45, 46, 47
 — dosage, Electr. 68
 — filtration of rays, Electr. 68
 — in treatment of cases of malignant disease (W. Hill), Laryng. 39
 — of case of cesophageal growth (N. S. Finzi and W. Hill), Clin. 160
 — of case of rodent ulcer (C. J. Symonds), Clin. 162
 — of excavating rodent ulcer of face (C. J. Symonds), Clin. 74
 — of malignant disease of neck (W. Hill), Laryng. 25
 — growth in neck (N. S. Finzi and W. Hill), Clin. 159
 — growths, action, Electr. 56
 — apparatus, Electr. 58
 — technique: external application, Electr. 59
 — internal application, Electr. 60
 — of recurrent epithelioma of nasal vestibules and adjacent areas after surgical removal (W. Hill), Laryng. 26
 — of sarcoma of ear and mastoid region (N. S. Finzi), Clin. 157
 — irradiation by, arguments for vaccinal hypothesis of, R.S.M. 156
 — treatment by, discussion on (Clin. 163-167): Dr. Iredell, Mr. Fedden, Mr. C. R. C. Lyster, Dr. Dunbar, Dr. Lazarus-Barlow, Mr. Eve, Mr. W. P. Herringham, Dr. Hill (reply), Dr. Finzi (reply)
 — by, relation to surgery and X-rays, Electr. 66
 Rainfall, effect on flies, Epid. 161
 — at Naples, Baln. 18, 19
 — in relation to prevalence of typhoid fever, Epid. 137
 Ramon y Cajal, development of neurone, quoted, Path. 4
 —, silver method of staining, Path. 22, 23
 — neuroblasts, Path. 11, 12
 Rats, inoculation with cat or mouse sarcoma, cannot protect against malignant disease of own species, Therap. 74
 — possible agency in spread of typhoid fever, Epid. 210
 Ravaut, lymphocytosis during secondary syphilis, Neur. 37
 Raw, N.—Discussion on diagnosis and treatment of duodenal ulcer, Surg. 111
 —, discussion on hospital infection of tuberculosis, Med. 160
 Raynaud's disease, similarity of condition of angioneurosis to, Clin. 107
 v. Recklinghausen, hæmorrhage, following chronic hypersecretion, Surg. 180
 Recklinghausen's disease, case of (E. G. Graham Little), Derm. 49
 — (F. Parkes Weber), Clin. 79

- Reconstruction model (wax-plate method of Born), showing auditory apparatus of 16 mm. human embryo (G. J. Jenkins), Otol. 34
- Rectal etherization, when to be employed, Anæsth. 71
- feeding in gastric ulcer, Therap. 135
- Rectum, continuous saline irrigation of (proctocolysis), Obst. 9
- , malignant growths, method of applying radium to, Electr. 61
- , passage of electrode through, for application to prostate, Electr. 142
- , prolapse of, treatment by static wave current, Electr. 107
- , stricture of, treatment by fibrolysin (H. C. Howard), Clin. 87
- Reflexes, abrogation under chloroform impossible, Anæsth. 65
- Refrigeration, electricity and cautery, treatment of nævi by (E. R. Morton), Electr. 37
- in treatment of chronic X-ray dermatitis, Electr. 44
- of lupus erythematosus and vulgaris, Electr. 44
- of moles, Electr. 44
- of nævi, Electr. 41
- of warts, Electr. 44
- Reichmann, gastric hypersecretion (gastro-succorrhœa), quoted, Surg. 177
- REID, A. D.—Discussion on relative value of various transformers, Electr. 157
- , discussion on X-ray treatment of lymphadenoma, Electr. 133
- Reid, G. Archdall, heredity in disease, alluded to, Obst. 79
- Remedies, proprietary, patent and secret; discussion opened by Professor W. E. Dixon, continued by Mr. Beddoes, Mr. Gamble, Dr. Duncanson, Dr. Martin, Mr. Woolcock, Mr. Harrison, Professor Cushny, Professor Dixon (reply), Therap. 81-100
- Renal dullness, Med. 229
- , diagnostic application, Med. 229
- Renaud, study of weight in measles, quoted, Epid. 52
- Reptilia, absence of trochanters in, Clin. 192
- Resonance (gastric nucleus), anatomical or descriptive value, Med. 234
- , causation, Med. 233
- , deformations, Med. 236
- , identification and delimitation, Med. 234
- , intra-thoracic not intra-abdominal when normal, Med. 233
- , percussion, Med. 239
- , percussion note, Med. 233
- , shape, Med. 232
- , situation, Med. 231
- , size, Med. 232
- , technique in determining, Med. 234
- , variations in size, Med. 235
- Resonator, see *Tambour*
- Respiration (abdominal), Laryng. 48
- (costal), Laryng. 48
- , failure of, cause of death in tetanus, Med. 40
- , under administration of anæsthetics, Anæsth. 60
- and heart action, failure of, under anæsthetic, associated with engorgement of thyroid gland, Anæsth. 45, 55
- , position of larynx during, Laryng. 48
- "Respirator-inhaler," Therap. 18
- Respiratory murmur, weakness of, on left side of chest, clinical indication of diagnosis of aneurysm of descending thoracic aorta, Clin. 126
- rotation of cricoid on thyroid cartilage, transverse axis of, in belly-breathing, illustrated by model (R. H. Scanes Spicer), Laryng. 41, 44, 49
- tract, upper catarrhal infections of, treatment by vaccine, difficult, R.S.M. 103
- Restraining bodies, Path. 128, 129
- Reverdin, rupture of tunica vaginalis in hydrocele, quoted, Surg. 166, 168, 169, 171, 172

- Rhabdomyoma of urinary bladder (S. G. Shattock), Path. 31
 ———, histology, Path. 33
 Rheinfelden, cure of female sterility at, explained, Baln. 10
 Rheumatism due to chronic suppurative lesions in mouth, treatment by vaccine therapy, R.S.M. 89, 90
 ———, importance of examination of teeth in, Odont. 95
 ——— in relation to endocarditis, Therap. 101, 102, 104, 114
 ——— to infective arthritis, Baln. 92
 ———, true, relation of certain cases of gonorrhœa to, Clin. 108
 ——— and spondylitis, with torticollis and subluxation (T. H. Openshaw), Clin. 37, 38
 ———, see also *Nodules, rheumatic*
 ——— (acute), heart-block in, Therap. 103
 ———, pericardial effusion, serous inflammatory, occurring in course of, Med. 56
 ———, treatment by salicylic acid and salicin, value proved, R.S.M. 109
 ——— (subacute), case of, treated by vaccine (Friedländer's bacillus), R.S.M. 177, 178
 ——— (muscular), supposed case of; *Bacillus pyocyaneus* in urine, R.S.M. 178
 Rheumatoid arthritis, see *Arthritis (rheumatoid)*
 Ribbert, H., malignant adenoma of liver, quoted, Path. 151, 152
 RICHARDS, H. MEREDITH.—Discussion on bovine tuberculous mastitis, Epid. 246
 ———, discussion on scarlet fever, home treatment and prevention, Epid. 70
 Rickets and lymphatism, Anæsth. 50, 53
 RIDOUT, C. A. S.—Case of extensive injury to the head in a boy aged 7, Child. 162
 ———, discussion on diagnosis and treatment of intussusception in children, Child. 175
 ———, intussusception of the small intestine, containing a sarcoma of intestinal wall; enterectomy; recovery, Child. 161
 Rieder-bismuth meal, Electr. 110
 Rieder, form of stomach after bismuth meal, Electr. 111
 Riegel, secretory activity in gastric disorders, Med. 26
 RIGBY, HUGH M.—Case of popliteal aneurysm in both legs cured by the Matas obliterative operation, Clin. 131; discussion (p. 132): Mr. H. M. Rigby, Mr. A. Pearce Gould
 Riggs's disease, Odont. 29
 RILOT, C. F.—Discussion on odontoceles and some other cysts, Odont. 160
 ———, discussion on result of extraction of six-year-old molars, Odont. 157
 ——— sterilizable film-holder, Odont. 89
 Ringworm affecting upper lip as well as beard region and hand in man (S. E. Dore), Derm. 111
 RITCHIE, Professor J.—Attenuation of virulence of micro-organisms in laboratories, quoted, Epid. 76
 Ritter, results of tuberculin administration in minute doses at Edmundsthal Heilstätte, Hamburg, R.S.M. 116
 ROBBINS, C.—Discussion on result of extraction of six-year-old molars, Odont. 157
 Roberts, D. Lloyd, fibroma of right tube, quoted, Obst. 239
 Roberts, Sir W., on rôle played by sodium salts in body, quoted, Baln. 73
 ———, respirator-inhaler, quoted, Therap. 18
 ROBINSON, H. BETHAM.—Case of ulcer of doubtful nature on the left arytenoid cartilage in a man aged 68, Laryng. 29; discussion (p. 29): Dr. Watson Williams, Mr. de Santi, Dr. Dundas Grant, Mr. Robinson (reply)
 ———, chronic laryngitis in a man aged 26? prolapse of ventricle on right side, Laryng. 30; discussion (p. 30): Dr. Dundas Grant, Dr. H. J. Davis, Mr. Herbert Tilley, Mr. Horsford, Dr. Jobson Horne, Mr. Robinson (reply)
 Rodent ulcer, case of (G. W. Dawson), Derm. 47
 ———, cystic, case of (J. M. H. MacLeod), Derm. 3
 ———, excavating, of face, under treatment by radium (C. J. Symonds), Clin. 74
 ———, treatment by electro-vaccination, R.S.M. 155
 ——— by radium (C. J. Symonds), Clin. 162
 ——— by Röntgen rays derived from static electricity, Electr. 81

- Rogers, Dr. L., use of hypertonic salt solutions in cholera, quoted, *Baln.* 74
- ROLLESTON, H. D.—Cirrhosis carcinomatosa, quoted, *Path.* 151
- , discussion on acholuric jaundice, *Clin.* 157
- on bluish pigmentation of skin, *Clin.* 142
- on case of angioneurosis, *Clin.* 106
- for diagnosis, *Clin.* 148
- of (?) hyperplastic tuberculosis of pelvic colon, *Clin.* 206
- of rheumatoid arthritis with albuminuria, and with great enlargement of liver, *Clin.* 62
- on combination of auscultatory and tactile methods of reading arterial pressure, *Med.* 210
- on pancreatic reaction and fecal analysis, *Med.* 203
- on primary hyperplastic tuberculosis of stomach and duodenum, *Clin.* 43
- on rheumatoid arthritis with enlargement of lymphatic glands and spleen, *Clin.* 19
- on spleno-medullary leukæmia, *Clin.* 138
- on thoracic aneurysm in a boy, *Clin.* 36
- , jaundice with areas of unpigmented skin, *Clin.* 195; discussion (p. 197): Dr. F. Parkes Weber, Dr. James Galloway, Mr. A. Pearce Gould, Dr. Rolleston (reply)
- , pigmentation of the circum-oral skin and of the buccal mucosa in pernicious anæmia, *Clin.* 9; discussion (p. 9): Dr. Parkes Weber, Sir Dyce Duckworth
- , sequel to a case of pigmentation of the circum-oral skin and of the buccal mucosa in pernicious anæmia, *Clin.* 216
- , and CARPENTER, G. H.—Scleroderma with sclerodactyly, *Clin.* 32; discussion (p. 34): Dr. J. Galloway, Dr. Herringham, Dr. Savill, Dr. Rolleston (reply)
- ROLLESTON, J. D.—Discussion on case of compression of trachea by enlarged thymus, *Child.* 47, 48
- , malignant diphtheria with multiple lesions in an infant, *Child.* 74
- , and MCCRICK, T.—Case of purpura fulminans, *Child.* 75
- Romberg's sign, presence of, in syphilitic disease of spinal cord, *Neur.* 56
- Röntgen-ray diagnosis, different apparatus used, *Electr.* 119
- Röntgen rays, action in leukæmia, *R.S.M.* 158
- , blood-changes after exposure to, *R.S.M.* 158
- , derived from static electricity, *Electr.* 81
- , in treatment of cases of lupus, molluscum contagiosum, and rodent ulcer, *Electr.* 81
- , determination of shape and outline of pericardium by, *Med.* 94
- , diagnosis of calculus, mistakes in, *Electr.* 96, 97
- , of malignant tumours of jaws, *Odont.* 158, 161
- , of pericardial effusion, *Med.* 63
- , of purulent pericardial effusion, *Med.* 93, 103
- , of renal calculus, *Electr.* 90
- , of rheumatoid arthritis and osteo-arthritis, *Baln.* 100, 102
- , of serous pericardial effusion, *Med.* 75
- , of ureteral calculus, *Electr.* 88
- , effect on glandular tumour of neck, *R.S.M.* 156
- , examination of intestines by (F. M. Groedel), *Electr.* 109
- , by, to discover position of calculus, *Electr.* 88
- , irradiation by, arguments for vaccinal hypothesis, *R.S.M.* 156
- , new pocket stereoscope for use with X-ray negatives (A. H. Pirie), *Electr.* 139
- , production of cytolytic or spermolytic serum by, *R.S.M.* 156
- , relative value of various types of high-tension transformers (including coil) used for (R. Morton), *Electr.* 149-160
- , protection from, *Electr.* 156
- , in treatment of enlarged glands in lymphadenoma (A. H. Pirie), *Electr.* 125-131
- , of fibroid tumours, *Obst.* 25

- Röntgen rays, relation of radium treatment to, *Electr.* 66
 ———, vaccinal action, *R.S.M.* 155
 Root-lesions in spinal syphilis, *Neur.* 57
 ROSE, F. A.—Discussion on case of "bleeding polypus" of the inferior turbinate, *Laryng.* 105
 ———, discussion on cast of upper jaw from a case of congenital occlusion of the posterior naris, *Laryng.* 115
 ——— on perforation of nasal septum from salt dust, *Laryng.* 5
 ——— on removal of a rapidly-growing soft fibroma from posterior wall of left maxillary antrum by a modified Rouge's operation, *Laryng.* 96
 ROSENBERG, negative results of sour-milk treatment, quoted, *Therap.* 61
 ROSENHEIM, O., and SHAW-MACKENZIE, J. A.—Lipoclastic accelerating action of serum as index in pathological conditions, *Path.* 168
 Rotch's sign (angle, notch), *Med.* 64, 73, 74, 78, 98
 ———, basic angle test, adjunct to, *Med.* 74
 ———, disappearance of in pericarditis, *Med.* 57, 86
 Rothschild villages in the Chilterns, *Baln.* 46
 Rotunda Hospital, statistics of placenta prævia, *Obst.* 168
 Rouge's operation in removal of nasal and ethmoidal endothelioma, *Laryng.* 67
 ——— modified in removal of rapidly growing soft fibroma from posterior wall of left maxillary antrum (H. Tilley), *Laryng.* 96
 ROUGHTON, E. W.—Two cases of hoarseness, *Laryng.* 40; discussion (p. 40): Dr. H. J. Davis, Mr. Tilley, Dr. FitzGerald Powell, Dr. Dundas Grant
 Round ligaments, Alexander's operation for shortening, in treatment of uncomplicated prolapse of uterus with retroflexion, *Obst.* 228
 ROUTH, AMAND.—Discussion on Cæsarean section in eclampsia gravidarum, *Obst.* 204
 ———, discussion on Cæsarean section in placenta prævia, *Obst.* 179
 ——— on case of cystic tumour of right broad ligament, *Obst.* 70
 ——— on fibro-adenoma of ovary, *Obst.* 52
 ——— on leucoplakic vulvitis and its relation to kraurosis vulvæ and carcinoma vulvæ, *Obst.* 49
 ——— on life-history of fibromyomata of uterus, *Obst.* 220
 ——— on malignant and innocent ovarian growths, *Obst.* 96
 ——— on points for the use of, and indications for, electrotherapy in some gynecological affections, *Obst.* 25
 Routh, C. H. F., advocacy of electrical treatment in gynecology, *Obst.* 27
 ROUX, serum-therapy of syphilis, quoted, *Surg.* 229
 ROWLANDS, R. P.—Discussion on case of (?) hyperplastic tuberculosis of pelvic colon, *Clin.* 207
 ———, see also *Clarke, S. A.*, case of phlegmonous gastritis
 ROWLETTE, ROBERT J.—Report on specimen of ovarian pregnancy (E. H. Tweedy), *Obst.* 132
 Rowntree, Seebohm, method of estimation of poverty in York, *Epid.* 4, 12, 31
 ——— and Sherwell, A., weekly expenditure on drink in working-man's family, *Epid.* 9
 Royal College of Surgeons of England, Museum specimens of rhabdomyoma of bladder in, *Path.* 31, 36, 38
 Royal Free Hospital, statistics of pericardial effusion from (Ivy E. Woodward), *Med.* 114
 Rubrospinal tracts, degeneration in cases of tumour of pineal body, *Neur.* 66, 78
 Rupia, mixed infection in, *R.S.M.* 20
 Rural districts, old age cause of pauperism in, *Epid.* 7
 RUSHTON, W.—Case of painful attrition, *Odont.* 118; discussion (p. 120): Mr. W. Hern, Mr. F. J. Bennett, Mr. J. F. Colyer, Mr. Gabell, Mr. S. Mummery, Mr. W. Rushton (reply)
 ——— discussion on the bacteriology of pyorrhœa alveolaris, *Odont.* 67
 ——— on result of extraction of six-year-old molars, *Odont.* 157

- RUSSELL, A. E.—Case of cyclic or recurrent vomiting associated with hypertrophic stenosis of the pylorus, *Child.* 78; discussion (p. 86): Dr. Langmead, Dr. Spriggs, Dr. Russell (reply)
- , discussion on heart massage in heart failure during anæsthesia, *Anæsth.* 13
- — on the treatment of tetanus, *Med.* 52
- Russell, Major, negative phase in opsonic index, *R.S.M.* 197
- Russians, castrating sect among (Skoptzi), *Path.* 103
- Sabouraud, pathology of acne, quoted, *Path.* 172
- Saccule, region of, surgical importance (S. R. Scott), *Otol.* 23
- Sacral percussion, *Med.* 290
- SAINSBURY, H.—Discussion on teaching of therapeutics in hospital wards, *Therap.* 9
- , discussion on treatment of endocarditis, *Therap.* 114
- St. Bartholomew's Hospital, mortality from tetanus at (1895-1907), *Med.* 39, 54
- —, statistics of pericardial effusion from, *Med.* 134
- Saint-Martin, experiments on strength of tunica vaginalis, quoted, *Surg.* 167
- , rupture of tunica vaginalis in acute hydrocele, quoted, *Surg.* 165, 167, 168, 169
- St. Mary's Hospital inoculation wards, methods employed and results obtained at, *R.S.M.* 16, 41, 129, 147
- St. Sauveur, cure of female sterility at, explained, *Baln.* 10
- St. Thomas's Hospital, statistics of pericardial effusion from (C. R. Box and G. G. Butler), *Med.* 104
- — Museum, specimen of rhabdomyoma of bladder in, *Path.* 39
- Sale of Food and Drugs Act (1875; 1899), *Therap.* 89
- Salicin in treatment of syphilis, *Surg.* 237
- Salicylate in treatment of rheumatism with endocarditis, *Therap.* 104
- Salicylic acid and salicin in treatment of acute rheumatism, value proved, *R.S.M.* 109
- Salies de Béarn, cure of female sterility at, explained, *Baln.* 10
- Saline solution, injections in control of vaccine treatment of whooping-cough, *R.S.M.* 99, 100
- —, subcutaneous injection in treatment of hæmorrhage, *Therap.* 123
- Saline waters of Llandrindod Wells, method of giving, *Baln.* 79
- —, proportion of salt in, to that contained in blood serum, *Baln.* 78, 79
- —, question of giving or withholding in cases of gout, *Baln.* 78, 79, 81, 84
- — (strong), purgative action of, *Baln.* 79
- — (weaker), diuretic action, *Baln.* 80, 84
- Salpingitis, treatment by electricity, *Electr.* 7, 8, 9, 13, 18, 20, 25
- (chronic), differential diagnosis from ectopic gestation difficult, *Obst.* 237
- (tuberculous), with tuberculous adenomyoma of Fallopian tube (J. Inglis Parsons and B. Glendining), *Obst.* 238
- Salt (common), avoidance in chronic nephritis, *Therap.* 143
- —, ingestion of, use and abuse in (R. Ackerley), *Baln.* 59-83
- —, see also under *Sodium chloride*
- Salt dust, perforation of nasal septum from (Dan McKenzie), *Laryng.* 3
- Salts, anhydrous, in dihydrogen sodium phosphate, *Med.* 16, 17
- Sanatoria in treatment of pulmonary tuberculosis combined with use of tuberculin, *R.S.M.* 116, 207
- — — — — of emulsion and other vaccines, *R.S.M.* 149, 150, 151
- — — — —, value proved, *R.S.M.* 109, 110
- Sanatorium accommodation in schools, *Epid.* 45
- Sanatorium treatment and galvano-cautery heal tuberculosis of epiglottis, left ary-epiglottic fold, and left inter-arytenoid space (StC. Thomson), *Laryng.* 7
- — and two months' silence heal tuberculosis of left vocal cord and inter-arytenoid space (StC. Thomson), *Laryng.* 6

- SANDILANDS, J. E.—The communication of diarrhoea from the sick to the healthy, *Epid.* 95-121; discussion (p. 122): Sir Shirley Murphy, Dr. Ralph Vincent, Dr. Sidney Davies, Mr. M. Greenwood, jun., Dr. Niven, Dr. Sandilands (reply), Dr. J. T. C. Nash (communication by letter)
- , discussion on summer diarrhoea and enteric fever, *Epid.* 269
- Sanitary requirements, compliance with, on part of recipients of public medical assistance advocated, *Epid.* 35
- Sansom, A. E., compression of left lower lobe of lung in pericarditis, quoted, *Med.* 84
- Sansom's pleximeter in delimitation of gastric nucleus of resonance, *Med.* 234
- DE SANTI, P. R. W.—Case of laryngeal growth, *Laryng.* 38; discussion (p. 38): Mr. Barwell, Dr. R. H. Hodgson, Dr. H. J. Davis, Mr. de Santi (reply)
- , discussion on case of probable malignant disease of larynx in woman, *Laryng.* 37
- on perforation of nasal septum from salt dust, *Laryng.* 4
- on specimen of larynx showing redevelopment of epithelioma on left cord after removal of right for same cause, September, 1896, *Laryng.* 34
- on thyrotomy for epithelioma of right vocal cord, *Laryng.* 18
- on ulcer of doubtful nature on the left arytenoid cartilage, *Laryng.* 29
- Sapremia, distinction from septicæmia, *Obst.* 223, 224, 231, 233, 234
- Sarcoma of the body of uterus from a girl aged 20 (H. Jellett), *Obst.* 186
- of bone, diagnosis from cystic disease, *Clin.* 195
- of ear and mastoid region, radium treatment (N. S. Finzi), *Clin.* 157
- , growth of, toxins render tissues unfavourable to, *Surg.* 12
- of humerus, diagnosis from cyst, *Child.* 99
- of ilium, treatment by Coley's fluid (A. Carless), *Clin.* 113
- of intestinal wall contained in intussusception of small intestine (C. A. S. Ridout), *Child.* 161
- of long bones, use of mixed toxins in, *Surg.* 12-16
- , resistance to implantation of carcinoma compatible with suitability for implantation of sarcoma, *Therap.* 76
- suggested by case of large chronic swelling in tonsillar region (W. Hill), *Laryng.* 132
- , treatment by mixed toxins, duration of, *Surg.* 18
- by radium, *Electr.* 66
- of uterus, when painful, *Obst.* 8
- , virulent, due to injury of bone, *Surg.* 11
- (inoperable), action of mixed toxins on, *Surg.* 9-12
- , treatment by bacterial toxins (W. B. Coley), *Surg.* 1-44
- — — — —, experimental proof, *Surg.* 2
- — — — —, final result with records and tables of cases, *Surg.* 24-42
- — — — —, history of method, *Surg.* 2
- — — — —, supported by clinical facts, *Surg.* 2
- — — — —, why cure results, *Surg.* 11
- — — — — with *Streptococcus erysipelas*, *Surg.* 4-6
- (primary), recurrence after operations for, mixed toxins as prophylactic against, *Surg.* 15
- (retroperitoneal) diagnosed as *Bacillus coli* infection, *R.S.M.* 142
- Sarcomatous growth contained in enteric intussusception, recovery, *Child.* 175
- simulated by myomatous tumour of uterus (H. Macnaughton-Jones), *Obst.* 103; report on specimen (C. Lockyer), *Obst.* 104
- tumour, enormous, in child aged 2 (H. B. Carlyll), *Child.* 154
- SARGENT, PERCY W. G.—Case of disappearing abdominal tumour (shown by Bernard A. Cheadle), *Clin.* 85
- SAUNDBY, R.—Discussion on rôle of fats in treatment of disorders of stomach, *Med.* 36
- SAUNDERS, P. W.—Myopathy (juvenile type), *Neur.* 100
- SAVILL, T. D.—Case of acroteric scleroderma, *Derm.* 33; discussion (p. 34): Dr. Colcott Fox, Dr. Pringle, Dr. Pernet, Dr. Savill (reply)

- SAVILL, T. D.—Discussion on cases of chronic syphilitic poliomyelitis and chronic meningo-myelitis, *Neur.* 26
- , discussion on cases of pruritus, *Derm.* 16
- , sequel of a case of scleroderma of the extremities (sclerodactylia, acroteric scleroderma of Hutchinson) associated with a previous and family history of angioneurotic oedema, *Clin.* 33; discussion (p. 34): Dr. J. Galloway, Dr. Herringham, Dr. Rolleston, Dr. Savill (reply)
- Scalp, case of lupus erythematosus almost exclusively limited to (Sir M. Morris), *Derm.* 98
- , detachment of entire (A. Pearce Gould), *Clin.* 129
- , lupus erythematosus of eleven years' duration localized to (J. H. Sequeira), *Derm.* 101
- Scar tissue, formation of, vaccine treatment of ulceration in place of, R.S.M. 14
- Scarlet fever, ambulances for conveyance of cases should be aseptic, *Epid.* 82
- , antiseptic application, *Epid.* 79
- , —, chief value in mild or recessive cases, *Epid.* 79
- , in babies, no isolation necessary, *Epid.* 64
- , complications, *Epid.* 88
- , control of (F. G. Crookshank), *Epid.* 73
- , Fiske memorial prize essay on, *Epid.* 86
- , limitation of seasonal prevalence by, *Epid.* 74
- , by removal of cases to isolation hospitals, *Epid.* 74, 80
- , duration of spread from case to case, *Epid.* 77, 89
- , epidemic, extent of normal cycle of, *Epid.* 77
- , fatal case of, demonstrating *Streptococcus conglomeratus* in situ: microscopical sections through mastoid antrum in (S. Scott), *Otol.* 18
- , home treatment and prevention (R. Milne), *Epid.* 57
- , and ichthyosis, *Epid.* 63
- , impure infection, bacteriological recognition, *Epid.* 80
- , —, tonsillar exudation, general sign of, *Epid.* 80
- , incidence in Manchester, *Epid.* 90, 91
- , intensive cases, *Epid.* 79, 86
- , inoculation in, *Epid.* 81, 85
- , —, by carbolic oil, *Epid.* 89
- , isolation unnecessary, *Epid.* 58
- , medical inspection, *Epid.* 63
- , mixture of cases of other infectious diseases with, *Epid.* 83, 89
- , natural history of, *Epid.* 74, 87, 88
- , and otorrhoea, *Epid.* 62
- , pencil infection in schools, *Epid.* 89
- , and pneumonia, *Epid.* 62
- , prevention of infection, new method, *Epid.* 57
- , pure infection, *Epid.* 80
- , recessive cases, *Epid.* 79, 86
- , reduction of occurrence-rate under bed-isolation system, *Epid.* 85
- , return cases, *Epid.* 76, 77
- , relapses, *Epid.* 93
- , sporadic, relation to dust, *Epid.* 78, 90
- , seasonal prevalence in Ireland, in early nineteenth century, *Epid.* 75, 88, 93
- , —, of, in London, date and period, *Epid.* 75
- , skin peeling in, how far infectious, *Epid.* 81
- , social condition of patients, *Epid.* 91
- , susceptibility to, *Epid.* 76, 78, 79, 86
- , tenacity of infection in schools, *Epid.* 90
- , traumatic and surgical cases, *Epid.* 86, 87, 93
- , treatment, question of, *Epid.* 88
- , virus of, attenuation, *Epid.* 78

- Scarlet fever, virus of, cases in which potency is greatest, *Epid.* 81, 82
 ———— extra-human phase of existence, *Epid.* 89, 90
 ————, future phases of, *Epid.* 77, 78, 91, 92
 ————, possibly in cattle, 77, 78, 91
 ————, intensification, *Epid.* 77
 ————, probability of destruction by strict aseptic hospital treatment, *Epid.* 80
 ————, reproduction of typical symptoms by, *Epid.* 76, 77
 ————, waves of infection, *Epid.* 89, 90
 ————, autumnal rise, *Epid.* 90, 91
 Scars, extensive acne with (Sir M. Morris and S. E. Dore), *Derm.* 7
 ———, multiple, simulating leucoderma syphiliticum (H. G. Adamson), *Derm.* 1
 SCHARLIEB, MARY A.—On the proportion of malignant to innocent ovarian growths, founded on a series of 150 cases, *Obst.* 85-95; discussion (p. 96): Dr. H. Macnaughton-Jones, Dr. Amand Routh, Mr. Glendining, Dr. Griffith, Mrs. Scharlieb (reply)
 Schauta, Professor, statistics of ovarian growths, quoted, *Obst.* 97, 98
 SCHELLING, C.—Case of fracture of the mandible set with a silver splint made by the castin process, *Odont.* 51
 ———, case of neuralgia due to impacted lower wisdom teeth, in which skiagrams helped the diagnosis, *Odont.* 114
 ———, discussion on suppurating dental cysts, invading antrum, *Odont.* 117
 ———, two cases of suppuration of the maxilla, *Odont.* 113
 Schinzach, cure of female sterility at, explained, *Baln.* 10
 Schlatter's disease, case of (G. Taylor), *Clin.* 20; (R. C. Elmslie), *Clin.* 189
 ———, see *Tibia*, also enlargement of tubercle of
 SCHOLEFIELD, R. E.—Discussion on laryngeal vertigo, *Laryng.* 80
 School children, medical examination of, *Epid.* 40
 Schools, infection of scarlet fever by use of pencils in, *Epid.* 89
 ———, scarlet fever in, tenacity of infection, *Epid.* 90
 Schottlaender, Professor, statistics of ovarian growths, quoted, *Obst.* 98
 Schreiber, gastric hypersecretion, quoted, *Surg.* 177
 Schröder, on pancreatic reaction, quoted, *Med.* 182
 SCHUSTER, EDGAR, and MOTT, F. W., F.R.S.—Examination of the brain of a man who lived seven hours after receiving a shock of 20,000 volts, *Path.* 140
 Sciatica, acute, treatment by faradaic current, injurious, *Electr.* 105
 ——— benefited by continuous current treatment, *Electr.* 105
 ———, indication for treatment by static wave current, *Electr.* 99, 100, 105, 106
 ———, treatment by fibrolysin, *Clin.* 89
 ——— by Morton wave current, *Electr.* 83, 84
 Sciatic nerve, regeneration after section, *Path.* 23
 Science, branches of, practical application of, not necessarily the province of experts in, *R.S.M.* 126
 ———, working knowledge valuable as well as expert, *R.S.M.* 127
 Scientific method, attitude of clinicians towards, *R.S.M.* 75, 76, 77
 Scirrhus of breast, examination of, *Path.* 164
 Sclerodactyly accompanying lupus erythematosus (J. H. Sequeira), *Derm.* 102
 ——— and acroteric scleroderma of Hutchinson associated with a previous family history of angioneurotic oedema (T. D. Savill), *Clin.* 33
 ——— and scleroderma (H. D. Rolleston and G. D. H. Carpenter), *Clin.* 32
 Scleroderma, acroteric, case of (T. D. Savill), *Derm.* 33
 ———, case for diagnosis (E. G. G. Little), *Derm.* 88
 ——— (circumscribed) with unusually numerous patches (E. G. Graham Little), *Derm.* 48
 ——— (diffuse), case of (E. G. Graham Little), *Derm.* 47
 ———, vomiting in, *Derm.* 47, 48
 ——— of extremities (sclerodactylia, acroteric scleroderma of Hutchinson) associated with a previous family history of angioneurotic oedema (T. D. Savill), *Clin.* 33

- Scleroderma, with sclerodactyly (H. D. Rolleston and G. D. H. Carpenter), Clin. 32
- Sclerosis, cerebellar, case of (G. Holmes), Neur. 89
- (disseminated), case of (W. J. Maloney for J. Taylor), Neur. 99
- (lateral), affection of posterior roots of lower cervical and upper dorsal nerves? tabes? (H. Campbell), Neur. 34
- Scoliosis (congenital), case (F. Langmead), Child. 130
- — —, cases tabulated, Child. 132
- Scopolamine, morphia, and chloroform, administration of, Obst. 9
- — —, administration to neurasthenics before operation, Anaesth. 67, 72
- SCOTT, S. GILBERT.—Discussion on relative value of various transformers, Electr. 159
- SCOTT, SYDNEY R.—Demonstration of some anatomical and pathological preparations, Otol. 22; discussion (p. 26): Mr. A. L. Whitehead, Dr. A. Bronner, Mr. C. H. Fagge, Mr. A. Cheate, Mr. Scott (reply)
- , discussion on auditory apparatus of 16 mm. human embryo, Otol. 35
- — — on cases of cerebral thrombosis, Neur. 86
- — — on hysterical deafness with active vestibular reactions, Otol. 74
- — — on pathological changes found in ear of deaf mutes, Otol. 13
- — — on pathology of cholesteatomata, Otol. 69
- , microscopical sections through the mastoid antrum in a fatal case of scarlet fever demonstrating *Streptococcus conglomeratus* in situ, Otol. 18
- Screaming, stomach-ache, and sickness in infants, common symptoms masking early onset of intussusception, Child. 167
- Screen, effect of violet-flame discharge on, Electr. 78
- Screen examination in diagnosis of calculus not wholly reliable, Electr. 96
- examinations under Snook's high-tension transformer, Electr. 158, 159
- Serofuloderma, treatment by tuberculin injections, cases (G. T. Western), Derm. 8
- Scrotum, elephantiasis of, Surg. 171
- , erysipelas of, Surg. 171
- Scurvy, pericardial effusion associated with, Med. 56
- (infantile), ? shortening of right humerus and left thigh due to unobserved fractures in early life (H. Burrows), Child. 165
- Seaports, casual labourers in, uncertain conditions of livelihood, Epid. 8
- Seasnake, common, see *Enhydrina valakadien*
- Seborrhœa, origin of, Path. 172
- Secretions (internal), defects in, suggested cause of adiposis dolorosa, Clin. 59
- — —, knowledge as to action as yet indefinite, Baln. 7
- — —, possible relation to determination of sex, Baln. 12
- — —, reciprocal action, important factor in maintenance of health, Baln. 11
- Secret remedies, Therap. 83, 84
- — —, patent and proprietary remedies, discussion on, opened by Professor W. E. Dixon, Therap. 82; discussion, renewed (pp. 91-100): Mr. Beddoes, Mr. Gamble, Dr. Duncanson, Dr. Martin, Mr. Woolcock, Mr. Harrison, Professor Cushny, Professor Dixon (reply)
- — —, published by British Medical Association, Therap. 95
- Seligmann, C. G., oöphorectomy, Path. 51, 52
- , on functions of spermatogenesis quoted, Path. 119
- and SHATTUCK, S. G.—The influence of oöphorectomy on the growth of the pelvis, Path. 102-126
- — —, and DUDGEON, L. S.—Attempts to produce chondromatous or osteomatous growths by the grafting of fetal bones (interim report), Path. 127-140
- SEMON, H. C. G., and CAMMIDGE, P. J.—An experimental investigation into the origin and cause of the pancreatic ("Cammidge") reaction in the urine, Path. 79-92
- Senator, atypic appendicitis, quoted, Surg. 187
- Senator's gelatine treatment of chronic gastric ulcer, Therap. 131
- Sense discharges, special, from organic disease (Sir W. R. Gowers: Hughlings Jackson lecture), Neur. 1

- Sense discharges, special, auditory, *Neur.* 5
 ————, olfactory, *Neur.* 2
 ————, visual, *Neur.* 11
- Sepsis, oral, results of, *Odont.* 3
- Septic infection in Cesarean section for puerperal eclampsia, *Obst.* 199
- Septicæmia, "arterial," *Therap.* 104
 ———, cases of, control of vaccine inoculation by opsonic index dispensed with, *R.S.M.* 33
 ———, distinction from sapræmia, *Obst.* 223, 224, 231, 233, 234
 ——— (general), case treated by vaccine inoculation, *R.S.M.* 59, 60
 ———, result of rupture in hydrocele, *Surg.* 167
 ——— of pyorrhœa alveolaris, *Odont.* 29
- Septum (nasal), extreme deflection of triangular cartilage, treated by septal fissure (L. H. Pegler), *Laryng.* 128
 ——— fracture of, complicating maxillary suppuration; operations; recovery (J. Donelan), *Laryng.* 89
 ———, perforation of, from salt (NaCl) dust (Dan McKenzie), *Laryng.* 3
 ———, submucoous resection in treatment of complete occlusion of nostril by deflected triangular cartilage in children, advisability discussed, *Laryng.* 56-58
 ——— in treatment of congenital occlusion of posterior nares, *Laryng.* 91, 92
 ———, risk of falling-in of bridge of nose, *Laryng.* 57, 58
 ———, syphilitic ulceration of, *Neur.* 51
- SEQUEIRA, J. H.—Case of *kératodermie blenorrhagique*, *Derm.* 77-80; discussion (p. 80): Dr. T. C. Fox, Dr. Whitfield, Dr. Sequeira (reply)
 ———, case of lupus disseminatus faciei, *Derm.* 61; discussion (p. 62): Dr. Pringle
 ——— of tertiary syphilis (gummatous teno-synovitis), *Derm.* 20
 ———, discussion on case of dermatitis artefacta, *Derm.* 29
 ——— of rodent ulcer, *Derm.* 5
 ———, lupus erythematosus associated with erythema iris, *Derm.* 94; discussion (p. 95): Sir Malcolm Morris, Dr. Pringle
 ——— of eleven years' duration localized to the scalp, *Derm.* 101
 ——— with epithelioma, *Derm.* 100
 ——— with sclerodactyly, *Derm.* 102
 ———, non-ulcerative tuberculides following lupus vulgaris, *Derm.* 52; discussion (p. 52): Dr. Pringle
 ———, symmetrical plantar nævoid tumours in an infant, *Derm.* 22; discussion (p. 22): Dr. Pringle
 ———, two cases of mycosis fungoides presenting quite different aspects of the disease, *Derm.* 35
 ———, an unusual case of lupus erythematosus, *Derm.* 62
 ——— and LITTLE, E. G. GRAHAM.—Case of pseudo-xanthoma elasticum, *Derm.* 69; discussion (p. 70): Dr. T. C. Fox
- Sequestrum, large mesial, exfoliation of, in case of destructive ulceration of large and soft palate (J. Dundas Grant), *Laryng.* 71
- Serositis, multiple, of tuberculous origin, improvement under cardiolysis (F. J. Poynton and W. Trotter), *Clin.* 199, 200
- Serous membranes, tuberculosis of, case of, relation of temperature to opsonic index in, *R.S.M.* 41, 42
- Serre, rupture of tunica vaginalis in hydrocele, quoted, *Surg.* 172
- Serum, action of, accelerated by lipoclastic as index of pathological conditions (J. A. Shaw-Mackenzie and O. Rosenheim), *Path.* 168
 ——— (anti-cancer), fallacy of, *Therap.* 72
 ——— (cytolytic or spermolytic), production by X-rays, *R.S.M.* 156
 ———, diagnosis, extension of method, *R.S.M.* 4
 ——— in pregnancy, *Obst.* 237
 ———, examination of, as guide to immunity, *R.S.M.* 74

- Serum, gravitation to diaphragmatic region dangerous, Obst. 8, 9
 — of healthy rabbits, effect of injections of various tuberculins and of tubercle endotoxin on opsonizing action of (R. T. Hewlett), Path. 165
 —, oral administration, R.S.M. 92, 94
 — in syphilitic patients, complement fixing powers of, Surg. 239
 Serum-test in diagnosis of syphilis, negative, largely, in primary stages, Surg. 220, 222
 — — —, positive in secondary and tertiary stages, Surg. 220
 Serum therapy of endocarditis, Therap. 104, 105, 114, 115
 — — — of syphilis attempted but fruitless, Surg. 229
 SEWELL, S. V., and TURNBULL, H. H.—Systematic histological examination of the central nervous system of a case of transverse lesion of the spinal cord in the lower cervical region, Path. 61-72
 Sewerage of Naples, Baln. 23
 Sex, determination of, possible relation of internal secretions to, Baln. 12
 Sex-incidence, equal in congenital syphilis, juvenile paralysis, and tabes, Neur. 43
 Sexual abuse, cause of kraurosis vulvæ, Obst. 38
 Sexual organs (female, accessory), hypoplasia following oöphorectomy, Path. 119
 — — — (male, accessory), hypoplasia following castration, Path. 119
 SHARP, ALEXANDER.—Case of audible tinnitus, Otol. 54; discussion (p. 54): Dr. Edward Law, Dr. H. J. Davis, Dr. Dundas Grant, Mr. Sharp (reply)
 SHATTOCK, S. G.—Amyloid degeneration in an adenoma of the liver, Path. 153
 —, large chylous cyst of the mesentery, Path. 98
 —, result of double oöphorectomy upon the growth of the uterus in the rabbit, Path. 51
 —, rhabdomyoma of the urinary bladder: vesicæ urinariæ tumor e telâ musculari transverse striatâ constructus (rhabdomyoma), Path. 31
 —, and SELIGMANN, C. G.—On function of spermatogenesis, quoted, Path. 119
 — — —, the influence of oöphorectomy on the growth of the pelvis, Path. 102-126
 — — —, and DUDGEON, L. S.—Attempts to produce chondromatous or osteomatous growths by the grafting of fetal bones (interim report), Path. 127-140
 SHAW, ERNEST H.—Case of actinomycosis of the ovary, Obst. 146
 —, squamous-celled carcinoma of a dermoid cyst of the ovary, Obst. 143; discussion (p. 145): Mr. Targett, Dr. Blacker
 — and FINZI, N. S.—Further report on a case of carcinoma of the breast treated by radium, Clin. 1; discussion (p. 3): Mr. Pearce Gould
 Shaw, L. E., and Perry, Sir E. C., hæmorrhage in duodenal ulcer, quoted, Surg. 75
 SHAW-MACKENZIE, J. A., and ROSENHEIM, O.—Lipoclastic accelerating action of serum as an index in pathological conditions, Path. 168
 Sheath-cells, part played by in regeneration of nerve-fibres, Neur. 24, 25, 26
 Sheep (short-legged), breed of, how produced, discussed, Path. 107
 Shellfish, consumption of, influence on spread of typhoid fever in Belfast, Epid. 270
 — — — — — in Manchester, Epid. 190, 191, 207-209
 —, propagation of typhoid fever by, Epid. 182, 183, 184, 189, 190
 Shelly, C. E., liability to infection during school life, Epid. 45, 46
 Shennan, channels of tuberculous infection, quoted, Child. 27, 28
 SHERREN, JAMES.—Meckel's diverticulum, containing calculi and producing colic, Clin. 11; discussion (p. 14): Mr. Carless, Mr. Pearce Gould, Dr. Garrod, Mr. Sherren (reply)
 Sherrington, C. S., regenerated nerve-fibres, quoted, Path. 12
 —, theory of "receptive substance" or "synapse," quoted, Path. 28
 Sherwell, A., and Rowntree, Seebohm, weekly expenditure on drink in working-man's family, Epid. 9
 Shock as cause of death in lymphatism, Med. 50
 — — — after operations for gastric cancer, Surg. 142
 Shoulder, dislocation, treatment by static wave current, Electr. 107
 Shoulder-joints (pyæmic), treated by tapping and incision (E. M. Corner and M. Huggins), Clin. 120

- SHUTTLEWORTH, G. E.—Discussion on ateliosis in a man, Child. 146
 —, discussion on case of achondroplasia in twin, and on case of unidactyly, Child. 42
 — — — — — of speech defect, Child. 129
 — — — — — of oxycephaly, Child. 128
 — — — — — on examination of blood-serum of idiots by Wassermann reaction, Neur. 122
 Sibson's notch, Med. 64
 — — — — —, disappearance of, in pericarditis, Med. 57
 Side-chain molecules thrown off by nerve elements, Neur. 48
 Sigmoid sinus, two cases of cholesteatoma of unusual size, extending into and causing obliteration of (G. Wilkinson), Otol. 82
 SILK, J. F.—Discussion on choice of anæsthetic, Anæsth. 75
 Silver method of Cajal, staining by, Path. 22, 23
 — — — — — nitrate, staining of neuroblasts with, Path. 11, 12
 Simmons, Dr., attack on proprietary medicines, quoted, Therap. 99
 Simms, H., bacteriology of pyorrhœa, quoted, Odont. 30, 32, 33
 SIMPSON, Professor W. J. R.—Discussion on bovine tuberculous mastitis, Epid. 247
 Sinclair, case-incidence in measles among telegraph messengers, quoted, Epid. 49
 SINCLAIR, Sir W. J.—The pelvic conditions resulting from the slighter forms of puerperal sepsis and their treatment, Obst. 221-231; discussion (p. 231): Dr. Herbert Spencer, Sir F. H. Champneys, Bart., Dr. Malins, Dr. Lewers, Dr. Eden, Dr. Macnaughton-Jones
 "Singer's node" on left cord (E. A. Peters), Laryng. 107
 Sinus, see *Frontal sinus*; *Sphenoidal sinus*
 Sinusitis, treatment by vaccine, R.S.M. 215
 Sinusoidal current, technique in gynæcological applications, Electr. 35
 Sister and brother, congenital enlargement of one limb, occurring in (O. L. Addison), Child. 43
 Skiagram, see *Radiography*
 Skin disease, certain cases benefited by lactic-acid administration, Therap. 54
 —, diseases of, benefited by reduction of salt in diet, Baln. 76, 77
 —, —, dietetic rules for patients suffering from, Baln. 77
 —, elimination by, see *Diaphoresis*
 —, functions of, Baln. 4
 —, —, see also *Pigmentation, dermic*
 —, inoculation of, protective value against cancer, Therap. 76
 —, liver and pancreas, pigmentary deposits in, in case of bronzed diabetes (H. S. French), Path. 94
 —, peeling in scarlet fever, how far infectious, Epid. 81
 —, pigmentation in (F. Parkes Weber and R. H. Norman), Clin. 140
 —, staphylococcic lesions of, treatment by vaccine, R.S.M. 188
 —, transmission of stimulation to internal secretory glands by, Baln. 4, 5
 —, tuberculosis of, see *Tuberculides*
 —, (unpigmented), jaundice with areas of, in case of cancer of cardiac orifice of stomach (H. D. Rolleston), Clin. 195
 Skoptzi (castrating Russian sect), Path. 103
 — — — — —, mutilations among females limited to external genitals (mamme and nipples), Path. 118
 — — — — —, stature of, Path. 105, 106
 Skull of Egyptian mummy, condition of teeth in, Odont. 154
 —, fracture of base of; operation; recovery (C. A. Ballance), Clin. 82
 — in male Belgian hare, effect of castration on, Path. 104, 105
 Skulls of dervishes, condition of teeth in, Odont. 154, 155
 — (Egyptian), exhibition of (A. S. Underwood), Odont. 153
 — — — — — exhibited, with front teeth mutilated, Odont. 153
 —, series of, demonstrating variations in the sphenoidal sinus and in the sphenothmoidal cell (W. S. Syme), Laryng. 37
 Skutsch's pelvimeter, Obst. 113, 121

- SLATER, CHARLES.—Discussion on vaccine therapy, R.S.M. 39
- Sleepiness, in cases of tumour of pineal body, *Neur.* 72
- SLOAN, S.—Discussion on electrical treatment of prostatic obstruction, *Electr.* 146
- on radium in treatment of malignant growths, *Electr.* 68
- on static wave current, *Electr.* 104, 107
- on therapeutical use of static electricity, *Electr.* 82
- on treatment of nævi by electricity, cautery, and refrigeration, *Electr.* 47
- on X-ray treatment of lymphadenoma, *Electr.* 132
- , electro-therapeutics in gynæcology: presidential address at opening meeting of Electro-Therapeutical Section, October 15, 1909, *Electr.* 1
- Smale and Colyer, on absorption of roots of permanent teeth, quoted, *Odont.* 14
- Smallpox, hæmorrhagic, diagnosis of purpura fulminans from, *Child.* 77
- SMITH, E. ARCHIBALD, and McDONAGH, J. E. R.—Case of pachydermatocele, *Clin.* 76
- Smith, C. N., on pancreatic reaction, quoted, *Med.* 182
- SMITH, E. BELLINGHAM.—Case of amaurotic family idiocy, *Child.* 148
- , discussion on malignant endocarditis of tricuspid valve, *Child.* 142
- Smith, G. Bellingham, bilateral cryptorchism, *Path.* 53
- SMITH, G. DARWALL.—Discussion on fibro-adenoma of ovary, *Obst.* 53
- SMITH, J. HENDERSON.—Discussion on vaccine therapy, R.S.M. 92
- SMITH, S. MAYNARD.—Case of congenital œdema of the leg, *Child.* 89; discussion (p. 89): Dr. Essex Wynter, Dr. F. Parkes Weber, Dr. Pernet
- , case of obliterative endaneurysmorrhaphy (Matas's operation) for popliteal aneurysm, *Clin.* 130
- , discussion on vaccine therapy, R.S.M. 128
- Smoking in causation of peculiar erythema of left palate (J. Dundas Grant), *Laryng.* 27
- SMYLY, Sir WILLIAM.—Discussion on Cæsarean section in placenta prævia, *Obst.* 13
- Snake-venom, poisoning by, acute degenerative changes in nervous system due to (W. K. Hunter), *Neur.* 105-115
- , intensity dependent upon time of action, rather than on amount injected, *Neur.* 108
- , shown by experiment, *Neur.* 106
- Snake-venoms, comparative toxicity, *Neur.* 116
- Snaring operation, hypertrophy of lingual tonsil, with impairment of singing voice, improved by (J. Dundas Grant), *Laryng.* 155
- "Snook" current, notification by spark-gap, *Electr.* 156, 157
- Snook's type of rectifying high-tension transformer, value of, in production of X-rays, *Electr.* 155-158
- Soamin, injection of, followed by blindness, *Surg.* 217, 225
- , in treatment of syphilis, *Surg.* 217, 225, 246
- Sodium, acid phosphate of, acidity of urine increased by, *Med.* 21
- , impurities in, *Med.* 16, 22, 24
- , solution of calcium-oxalate calculus by: by laboratory experiment, *Med.* 13-20
- , in urinary passages, *Med.* 11-13, 21
- Sodium chloride, abstinence from, presumed immunity from cholera due to, *Baln.* 61
- , effects on solubility of sodium biurate, *Baln.* 73
- , excretion of, from body, *Baln.* 66
- , general channels for, *Baln.* 70
- , by the kidneys, *Baln.* 67, 69
- , hypertonic solutions of, use in cholera, *Baln.* 74
- , ingestion of, abnormal, *Baln.* 75
- , or abstinence from, effect upon horses and cattle, *Baln.* 61, 62
- , an acquired or educated taste, *Baln.* 62, 63
- , causing reduction in obesity, *Baln.* 75
- , excess in, *Baln.* 64
- , causation of gout by, *Baln.* 73, 74, 75

- Sodium chloride, ingestion of, excess in, causing eczema, Baln. 76, 77
 ———— causing œdema, Baln. 75
 ———— in cooked foods, Baln. 65, 66
 ————, ill-effects of, Baln. 65
 ———— ———— on children, Baln. 71
 ———— ————, increase of blood-pressure due to, Baln. 74
 ————, practice general, but not universal, Baln. 60
 ————, reduction, benefit in skin disease, Baln. 76, 77
 ————, use and abuse in (R. Ackerley), Baln. 59-83
 ————, percentage of, in fluids, tissues and organs of body, Baln. 74
 ————, proportion in saline waters compared with that contained in blood-serum, Baln. 78, 79
 ————, purgative qualities of, Baln. 84
 ————, retention longer and excretion slower after ingestion with food than when taken with water, Baln. 79
 ———— in urine, method of estimation, Baln. 82. See also *Salt (common)*
 ————, phosphate of (acid), precipitation of calcium oxalate in urine prevented by, Med. 2
 ———— phosphates in urine, determination by uranium nitrate, Med. 15
 ———— salicylate and bicarbonate in treatment of rheumatic pericarditis, Med. 71
 ———— in treatment of rheumatic pericarditis, Med. 71
 Soil, growth of pathogenic bacilli in, Epid. 138, 139
 Soles, see *Palms and soles*
 SOLL, E.—Discussion on arthritis deformans, Baln. 97
 ————, discussion on saline waters and common salt, Baln. 83
 Sour-milk treatment, arguments against, Therap. 60, 61
 ————, fashion or craze for, Therap. 51, 58, 59, 61
 ————, selection of cases for, Therap. 59
 Southard, organic disease cause of olfactory discharges, Neur. 2, 4
 SPANTON, W. D.—Discussion on Cæsarean section in eclampsia gravidarum, Obst. 205
 ————, discussion on electrotherapy in some gynæcological affections, Obst. 25
 ————, remarks on delivery of presidential address (pain associated with disease of genital organs, Obst. 10
 Spark-application in static electricity, prevention of long flashes, Electr. 80
 ———— in treatment of lupus, Electr. 78-80, 84
 ————, cause of curative effect, Electr. 79, 84
 ————, technique, Electr. 79
 ———— of telangiectatic facial nævus, Electr. 80
 ————, see also *Brush discharges*
 Spark-gap, notification of "Snook" current by, Electr. 156, 157
 Spasm (flexor), case of (G. A. Sutherland), Clin. 19
 ———— (muscular), in hypertrophic stenosis of pylorus, Child. 84, 85
 ————, factor in causation of recurrent or cyclic vomiting, Child. 85
 ———— (pyloric), symptom of appendicitis, Surg. 187, 201, 205, 207
 Spasms, relief of, in tetanus, Med. 41
 Speech defect, case of (R. Hutchison), Child. 129
 SPENCER, H. R.—Discussion on abdominal hysterectomy for necrosed and suppurating sub-peritoneal fibroid, Obst. 65
 ————, discussion on Cæsarean section in eclampsia gravidarum, Obst. 204
 ———— in placenta prævia, Obst. 177
 ———— on case of myomectomy during pregnancy, Obst. 166
 ———— of primary carcinoma of Fallopian tube, Obst. 78
 ———— on fibro-adenoma of ovary, Obst. 52
 ———— on life-history of fibromyomata of uterus, Obst. 220
 ———— on modern methods of delivery in contracted pelvis, Obst. 123
 ———— on slighter forms of puerperal sepsis, Obst. 231

- SPENCER, H. R.—Posterior Cesarean section, followed by total hysterectomy, for fibroids, Obst. 82
- SPENCER, W. G.—Case of trephining for general epilepsy after an interval of three years, Clin. 65
- , detachment of the tendon of the subscapularis muscle for recurring dislocation of the humerus, Clin. 20
- , discussion on case of excision of both maxillæ, Clin. 11
- on radical operation for cancer of pylorus, Surg. 160
- on surgery of malignant disease of the tongue, Clin. 186
- , olfactory discharges as warning in epilepsy, quoted, Neur. 4
- Spermatogenesis, function of, remote effects, Path. 119
- Sphenoidal sinus and sphenoidal cell, variations in relations of, demonstrated by series of skulls (W. S. Syme), Laryng. 37
- Sphygmomanometer, accuracy of record, not portability, essential, R.S.M. 76
- SPICER, R. H. SCANES.—Case of intranasal disease (obstruction, pressure, and ethmoidal suppuration), associated with aggravated, lifelong stammering, Laryng. 119
- , case of œsophageal stenosis in a man aged 53, Laryng. 117; discussion (p. 118); Dr. Finzi
- , discussion on bands between Eustachian tube and pharynx, Laryng. 70
- on case of asthma; improvement after nasal treatment, Laryng. 116
- of laryngeal new growth for diagnosis, Laryng. 61
- of tuberculosis of pharynx, Laryng. 59
- on diseases of the ventricle of the larynx, Laryng. 158
- on endothelioma of ethmoid, Laryng. 66
- on extrinsic carcinoma of larynx, Laryng. 54
- on globular dilatation of œsophagus, Laryng. 165
- on growth in nasopharynx resembling pale adenoids, Laryng. 73
- on hypertrophy of lingual tonsil, Laryng. 156
- on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 57
- on removal of penny from gullet, Laryng. 135
- on sanatorium, silence, and galvano-cautery in treatment of tuberculosis of throat, Laryng. 9
- on thyrotomy for epithelioma of right vocal cord, Laryng. 18
- on treatment of deflection of triangular cartilage by septal fissure, Laryng. 129
- of tuberculous laryngitis by galvano-puncture, Laryng. 122
- on tuberculosis of larynx in girl aged 12, Laryng. 127
- on vulsellum-catch forceps, Laryng. 125
- , model by which the variation in effect of "back" and "belly" breathing on the stresses, strains, and frictions in the throat and larynx, more especially of the cricoid cartilage on the spinal column, and also the transverse axis of respiratory rotation of the cricoid on the thyroid cartilage, can be illustrated, Laryng. 41; discussion (pp. 48-52): Dr. H. J. Davis, Dr. Harry Campbell, Mr. Cresswell Baber, Mr. Horsford, Dr. Halls Dally, Dr. Dundas Grant, Dr. Jobson Horne, Dr. Donelan, Dr. Scanes Spicer (reply)
- SPILSBURY, B.—Discussion on lymphatism, Anæsth. 39
- , and HARRIS, WILFRED.—Acute cerebral softening? due to venous thrombosis, Neur. 130; discussion (p. 132): Dr. F. E. Batten, Dr. Farquhar Buzzard, Dr. Trevelyan, Dr. Harris (reply)
- Spinal band of Piorry, Med. 221
- Spinal canal, retroperitoneal teratoma connected with (R. Johnson and T. W. P. Lawrence), Path. 93
- Spinal column, relation of cricoid cartilage to, in back and belly breathing (R. H. Scanes Spicer), Laryng. 41
- Spinal cord, chromatolysis of cells of, from case of electric shock, Path. 145

- Spinal cord, compression of, with chronic syphilitic meningitis (T. Grainger Stewart), Neur. 33
- , —, see also *Paraplegia (compression)*
- , —, dorsal columns, how far affected in cases of spinal syphilis, Neur. 61
- , —, irritative lesions in, as a cause of changes peculiar to rheumatoid arthritis, Baln. 103, 106
- , —, peripheral tracts, degeneration of, in cases of spinal syphilis, Neur. 60, 61
- , —, sclerosis of, disseminated, diagnosis from disseminated syphilis, Neur. 63, 64
- , —, soft membranes, thickening of, in cases of spinal syphilis, Neur. 60
- , —, syphilis of, latent, Neur. 37, 38
- , —, transverse lesion of, in lower cervical region, histological examination of central nervous system in case of (S. V. Sewell and H. H. Turnbull), Path. 61
- , —, —, areas of degeneration, Path. 70
- Spindles of von Ebner (D. E. Coysh), Odont. 144
- Spine, fracture-dislocation in mid-cervical region resulting in transverse lesion in lower cervical region of cord (S. V. Sewell and H. H. Turnbull), Path. 61
- , —, malformation of, causing congenital scoliosis, Child. 131
- Spinous processes, whole series of, percussion values, Med. 214
- Spirilla*, Odont. 31
- Spirits Act, Therap. 89
- Spirochæta, species of, demonstration by ultra-microscope (H. W. Bayly), Clin. 3
- , —, points of distinction from *Treponema pallidum*, Clin. 4
- Spirochæta pallida*, causation of syphilitic cutaneous eruption by, Med. 36
- , —, demonstration of presence in human ovum, Neur. 46
- , —, discovery of, Surg. 210
- , —, presence in syphilis, Surg. 222
- , —, —, combined with other micro-organisms, Surg. 237
- , —, —, methods of detection, Surg. 219, 238
- , —, specimen of, prepared with Indian ink, with demonstration of method (J. E. R. McDonagh), Derm. 73
- Spitta, H. J., oral administration of tuberculin, quoted, R.S.M. 118
- Spleen, enlarged, case of (V. Dickinson), Child. 55
- , —, treatment by static wave current, Electr. 103
- , —, and lymphatic glands, enlargement of, in rheumatoid arthritis (E. I. Spriggs), Clin. 15
- , —, tissue, inoculation of, protective value against epithelial tumours, Therap. 76
- Splenic dullness as depicted by Piorry, Med. 217
- , —, diagnostic applications, Med. 228
- SPOKES, S.—Discussion on condition of the mouth in chronic disease, Odont. 110
- Spondylitis, rheumatic, with torticollis and subluxation (T. H. Openshaw), Clin. 37, 38
- Sprains, treatment by static induced current, Electr. 102, 103
- SPRIGGS, E. I.—After-results of cases treated by Lenhartz method, quoted, Therap. 131
- , —, case of acromegaly, Clin. 149
- , —, of rheumatoid arthritis with enlargement of lymphatic glands and spleen, Clin. 15; discussion (p. 18): Dr. Garrod, Dr. Rolleston, Sir Dyce Duckworth, Dr. Spriggs (reply)
- , —, congenital thoracic deformity, absence of part of the pectoralis major, Child. 102; discussion (p. 102): Dr. E. Cautley
- , —, discussion on case of chloroma, Child. 20
- , —, —, for diagnosis, Child. 24
- , —, —, of multiple peripheral neuritis in a child, Child. 40
- , —, on criticism of some principles in treatment of chronic nephritis, Therap. 147
- , —, on cyclic or recurrent vomiting, Child. 87
- , —, on extensive myositis ossificans, Clin. 154
- , —, on specimen of heart of infant showing nodules on endocardium, Child. 46
- , —, on treatment of gastric ulcer, Therap. 133
- , —, on use of citrated milk for wasting infants, Child. 123

- SPRIGGS, E. I., and FIRTH, A. C. D.—Congenital malformation of heart showing complete atresia of the pulmonary artery, *Child.* 49; discussion (p. 50): Dr. J. Porter Parkinson
- and HUGO, H. F. L.—X-ray photographs from a boy, aged 3, the subject of habitual constipation, *Child.* 64; discussion (p. 66): Dr. Eric Pritchard, Dr. Milner Burgess, Mr. Lockhart Mummery
- Sprue, chronic pancreatitis in, *Med.* 172
- Sputum, cultivation of organism from, to procure autogenous pneumococcus vaccine, *R.S.M.* 166
- SQUIRE, J. EDWARD, C.B.—Hospital infection of tuberculosis as exemplified by the records of the resident staff of the Mount Vernon Hospital for Consumption and Diseases of the Chest for the past fifteen years, *Med.* 137-156; discussion (p. 157): Dr. Theodore Williams, Sir S. Murphy, Professor Osler, Dr. Heron, Dr. N. Raw, Dr. Wethered, Dr. Squire (reply)
- Stammering (life-long), case of, associated with intranasal disease (obstruction, pressure, and ethmoidal suppuration) (R. H. Scanes Spicer), *Laryng.* 119
- Staphylococcus infection, treatment by vaccine, *R.S.M.* 67, 114
- , opsonic index to, determination easy, *R.S.M.* 121
- (secondary), in case of tuberculosis of glands of neck, recovery after treatment with tuberculin, *R.S.M.* 115
- of skin, see *Boils*
- , invasion in cancer, *R.S.M.* 18
- , presence in cases of renal and ureteral calculus, *R.S.M.* 16, 17
- present in cultures from hay fever, *R.S.M.* 16
- , in lesions of pruritus ani, *R.S.M.* 15
- vaccine, dosage of, *R.S.M.* 96
- , in treatment of acne vulgaris, *R.S.M.* 137
- of glycosuria, *R.S.M.* 18, 178-181
- of pyorrhœa alveolaris, *Odont.* 43
- of staphylococcal skin lesions, *R.S.M.* 188
- (autogenous), injections of, in treatment of staphylococcal infection of lung, favourable, *R.S.M.* 205
- and streptococcus, mixed infection of, in rupia, *R.S.M.* 20
- Staphylococcus albus*, *Odont.* 30, 31, 33
- *aureus*, presence in lesions following mycosis fungoides, *Clin.* 8
- in pyorrhœa alveolaris, *R.S.M.* 173
- *pyogenes albus*, *Path.* 172
- *aureus*, *Odont.* 30, 31, 32, 36, 37, 39, 43
- STARLING, E. A.—Discussion on heart massage in heart failure during anaesthesia, *Anæsth.* 14
- Starvation (acute) accompanies recurrent or cyclic vomiting, *Child.* 82
- , fatty degeneration of liver in, *Child.* 82
- , relation to acidosis, *Child.* 82
- Static bath, use of, *Electr.* 104
- electricity in treatment of acute prostatitis following gonorrhœa, *Electr.* 148
- induced current, *Electr.* 104
- , applications, *Electr.* 105
- , description of, *Electr.* 108
- , treatment by, indicated in obstinate cases, *Electr.* 102
- Static machine, efficiency of, necessity for, *Electr.* 97, 98
- , value of, in production of X-rays discussed, *Electr.* 150
- Static wave current (F. H. Humphris), *Electr.* 97, 104, 108
- , duration of application, *Electr.* 99
- , indications for treatment by, *Electr.* 99-104, 104-108
- , technique, *Electr.* 98, 105
- , in treatment of dysmenorrhœa, *Electr.* 101

- Stomach, cancer of, symptoms, duration, Surg. 133
 ———, treatment by exploratory operation, Surg. 136
 ——— by operative methods, Surg. 140
 ———, mortality after, Surg. 160, 163
 ———, reason for failures, Surg. 118
 ——— by radical operation, limits to and contra-indications for, Surg. 145
 ———, remote results, Surg. 155, 156
 ——— in two stages, cases illustrating, Surg. 142-144
 ———, objections to, Surg. 160, 161
 ———, with removal of lymph area, Surg. 147-152
 ———, see also *Pylorus*
 ———, dilatation, Med. 235
 ———, distension of, in early phthisis, R.S.M. 17
 ——— in cases of cancer, Surg. 135
 ——— in duodenal ulcer, Surg. 73
 ———, with dyspepsia, relieved by static wave current, Electr. 104
 ——— and ptosis combined, Med. 235
 ——— symptoms, Surg. 185
 ———, upward, various types as revealed by percussion, Med. 236
 ———, diseases of, diagnosis by dorsal percussion, Med. 239
 ———, rôle of fats in treatment of (F. C. Moore and R. L. Ferguson), Med. 25
 ———, dorsal percussion of (W. Ewart), Med. 211
 ——— and duodenum, division of, sixth step in operation for removal of lymphatic area connected with stomach, Surg. 153
 ———, primary hyperplastic tuberculosis of (W. G. Nash), Clin. 40
 ———, examination of, by Röntgen rays, Electr. 109
 ———, hæmorrhage from, diagnosis, Surg. 96
 ———, omentum largest lymph-bearing area connected with, Surg. 147
 ———, operations on, in cases of mistaken diagnosis of malignancy, Surg. 138
 ———, percussion of, dorsal, Med. 231
 ———, ptosis of, Med. 235
 ———, resection of, see *Gastrectomy*
 ———, tumour of, disappearance, no proof of benignity, Surg. 139
 ———, treatment, operative in two stages, Surg. 139
 ———, ulcer of, acute action of hyperacidity of gastric juice in producing, Therap. 124, 126, 132, 137
 ——— (acute), age and sex incidence, Therap. 120
 ——— and chronic, gradations between, Therap. 120, 134
 ———, symptoms common to both forms, Therap. 119
 ———, distinction from chronic, Therap. 118, 119, 134
 ———, healing of, Therap. 125
 ———, hæmorrhage in, treatment, Therap. 121, 123
 ———, treatment, Therap. 120
 ———, dietetic, Therap. 126, 127, 134
 ———, operative, Therap. 124
 ———, anemia in, Therap. 135, 136, 137
 ———, appendicular gastralgia diagnosed as, Surg. 203, 204
 ——— and cancer, relation between, from histological point of view, Surg. 137
 ——— (chronic), association with arterio-sclerosis, Therap. 134
 ———, pancreatitis in, Med. 172
 ———, treatment, dietetic, Therap. 128, 130, 134
 ———, operative, Therap. 129, 131
 ———, prophylactic, Therap. 132
 ———, vomiting in, treatment, Therap. 130
 ———, co-existent with duodenal ulcer, Surg. 93, 94

- Stomach, ulcer of, diagnosis, difficulty of, Therap. 118, 135
 ———, fats in treatment of, Med. 32, 34
 ——— and gastric hypersecretion, Surg. 180, 181, 182, 184, 185, 186
 ———, method of feeding in, Med. 122, 134, 135
 ———, rectal, Therap. 135
 ———, mistaken for duodenal ulcer, Surg. 93, 94
 ———, multiple, with one only malignant, Surg. 139
 ———, site of origin, Therap. 135
 ———, treatment (C. Bolton), Therap. 117-133
 ———, vomiting of complete mucous membrane of pyloric half in case of phlegmonous gastritis from hydrochloric acid poisoning (S. A. Clarke, A. F. Hertz, and R. P. Rowlands), Clin. 209
- Stomach-ache, screaming and sickness in infants, common symptoms masking early onset of intussusception, Child. 167
- Stomatitis, ulcerative, result of pyorrhœa alveolaris, Odont. 29
- Stonham, C., report of a case of heart failure during anaesthesia, quoted, Anæsth. 8
- STOWERS, J. H.—Case of chronic tuberculosis cutis, Derm. 53; discussion (p. 54): Mr. Dawson, Dr. Stowers (reply)
 ———, discussion on cases of urticaria, Derm. 47
 ——— on scleroderma of diffuse type, Derm. 48
- Strangeways, T. P., measurement of opsonic index, R.S.M. 26
- Strasburger's fermentation process in examination of feces, Therap. 55, 56, 57
- Strassmann, tuberculous infection of tonsils, quoted, Child. 28
- Strockeisen, Cesarean section in eclampsia, Obst. 196, 197, 198, 200
- Streptococcus, agglutination of, in case of malignant endocarditis, R.S.M. 118
 ——— infecting operation-wound in breast, R.S.M. 69
 ——— infection, localized, vaccine inoculations not controlled by opsonic index, R.S.M. 32
 ———, opsonic index to, R.S.M. 121
 ——— serum injections in treatment of streptococcal infection of lung, result unfavourable, R.S.M. 205
 ——— vaccine, inoculation in case of streptococcal infection of middle ear, R.S.M. 68
 ———, oral administration in treatment of case of malignant endocarditis, R.S.M. 117, 118
 ——— in treatment of streptococcal infection of mouth, R.S.M. 88, Odont. 45
 ——— (autogenous or stock) in treatment of pyorrhœa alveolaris, R.S.M. 174
 ——— and staphylococcus, mixed infection of, in rupia, R.S.M. 20
- Streptococcus anginosus*, Otol. 20
 ——— *aureus*, cultivated from urine in case of puerperal septicæmia, R.S.M. 59
 ——— *brevis*, Odont. 30, 31, 33
 ——— *conglomeratus*, demonstrated *in situ*, in fatal case of scarlet fever, by microscopical sections through mastoid antrum (S. R. Scott), Otol. 81
 ———, differentiation from other species, Otol. 20
 ——— *erysipelas* broth, method of preparation, Surg. 8
 ———, treatment of inoperable carcinoma and sarcoma with, Surg. 4-6
 ——— and *Bacillus prodigiosus*, mixed toxins of, in treatment of inoperable sarcoma (W. B. Coley), Surg. 1-44
 ———, see also *Bacillus prodigiosus*
 ——— *fecalis*, Odont. 31
 ———, mouth organism identified as, R.S.M. 88
 ———, intestinal invasion by, effect of lactic bacillus on, Therap. 60, 62
 ——— *lanceolatus*, Odont. 33, 36, 37, 40
 ———, presence in empyema, R.S.M. 58
 ——— *mucosus*, culture from pleural cavity in case of pneumonia, R.S.M. 171
 ——— *pyogenes*, Otol. 20
 ———, cultivated from case of general septicæmia, R.S.M. 60

- Streptococcus pyogenes longus*, Odont. 30, 33, 36, 37, 39, 41, 45, 46
 ———, presence in abscess of liver, R.S.M. 57
 ——— *scarlatinae*, Otol. 20
 Strychnine, administration of, preceding scopolamine and morphine, not recommended, Anæsth. 67
 ———, method of administration in treatment of paresis of palate, pharynx, and œsophagus, Laryng. 90, 91
 ———, poisoning by, effect of intraspinal (intrathecal) injection of cocaine on, shown experimentally, Med. 52
 STUART-LOW, W.—Cases of extra-laryngeal inoperable carcinoma, shown to illustrate the beneficial effects of operation on the thyroid gland, Laryng. 13; discussion (p. 16): Dr. Dundas Grant, Dr. Donelan, Mr. Stuart-Low (reply)
 ———, discussion on endothelioma of ethmoid, Laryng. 65
 ——— on question of operation for complete occlusion of left nostril by deflected triangular cartilage in child, Laryng. 56
 ——— on treatment of deflection of triangular cartilage by septal fissure, Laryng. 129
 ——— on vulsellum-catch forceps, Laryng. 125
 ———, loss of voice, with dyspnoea, in a woman aged 26, Laryng. 162; discussion (p. 163): Dr. J. Dundas Grant, Mr. Stuart-Low (reply)
 ———, lupus of the nose, hard palate, fauces and epiglottis, in a girl aged 10, Laryng. 86; discussion (p. 87): Dr. Vinrace, Dr. Kelson, Mr. Barwell, Mr. Stuart-Low (reply)
 Sturges, O., percussion signs of amount of pericardial effusion, Med. 106
 ———, rheumatic pericarditis, quoted, Med. 75, 100
 STURRIDGE, E.—Discussion on bacteriology of pyorrhœa alveolaris, Odont. 62
 ——— discussion on condition of the mouth in chronic disease, Odont. 110
 ———, discussion on unilateral overgrowth of lower jaw, Odont. 49
 Subcutaneous injection of magnesium-sulphate solution in treatment of tetanus, Med. 50
 Subinvolution, uterine, treatment by electricity, Electr. 7, 8, 11, 22
 Subluxation, alto-axoid, and torticollis, with rheumatic spondylitis (T. H. Openshaw), Clin. 38
 ——— and torticollis, with rheumatic spondylitis (T. H. Openshaw), Clin. 37
 Subscapularis muscle, tendon of, detachment, for recurring dislocation of humerus (W. G. Spencer), Clin. 20
 Succinic acid, Med. 6
 Suchier, therapeutic effect of violet-flame discharge in static electricity, quoted, Electr. 77
 Suchier's method of treatment by static electricity, Electr. 73, 74
 Südmersen, pathology of acne, quoted, Path. 173
 Summer diarrhœa, annual wave of, explanation, Epid. 138
 ———, definition, Epid. 132
 ———, distribution among infants, Epid. 136
 ———, fatal cases in relation to prevalence of house-flies, Epid. 145-149, 270
 ———, infectious, Epid. 133
 ——— in Manchester, Epid. 134-136
 ———, death-rate compared with temperature and rainfall, Epid. 167, 168
 ———, deaths from, 1904-9, Epid. 149-152; compared with onset of typhoid-fever cases 1891-1906, Epid. 192-206
 ———, fatal cases in relation to prevalence of flies, Epid. 169
 ———, micro-organisms producing, Epid. 133
 ———, relation of cases and deaths to temperatures, Epid. 153
 ——— of privy-middens to, Epid. 134
 ———, seasonal prevalence, cause of, Epid. 137
 ———, transmission of, alleged factors, Epid. 138-141
 ——— by house-flies, Epid. 157-159, 266, 267
 ——— in Wigan, Epid. 259
 Sun, influence on dermic pigmentation, Baln. 4

- SUNDERLAND, S.—Discussion on arthritis deformans, *Baln.* 111
- Sundew, reflex action of, *Path.* 29
- Sun-spots, influence of, on atmospheric electricity, *Baln.* 54
- Suppuration (aural), case of long-continued, with spontaneous cure? (G. N. Biggs), *Otol.* 15
- Suprahepatic line, *Med.* 226
- Suprarenal capsules, fibro-caseous disease (extensive but incomplete) of both, with absence of symptoms of Addison's disease (H. French), *Path.* 93
- glands, disturbance in Addison's disease, cause of dermic pigmentation, *Derm.* 11
- Surgery (general), relation of dentistry to, *Odont.* 4
- of radium therapy to, *Electr.* 66
- von Sury, on question of thymus death, quoted, *Child.* 47
- SUTHERLAND, G. A.—Case of flexor spasm, *Clin.* 19
- , discussion on case of blue patches, *Child.* 133
- case of chloroma, *Child.* 20
- of cirrhosis of liver, *Child.* 43
- of multiple peripheral neuritis in a child, *Child.* 40
- of pneumococcal infection in infant simulating generalized tuberculosis, *Child.* 16
- on cases of oxycephaly, *Child.* 127
- SWAINSON, J. M. G.—Strangulated ovarian tumour in a child: operation, recovery, *Child.* 72
- Swallowing, difficulty in, means of relieving, *Laryng.* 153
- SYME, W. S.—Series of skulls demonstrating variations in the relations of the sphenoidal sinus and of the sphenoid-ethmoidal cell, *Laryng.* 37
- Syme's method of operation in malignant disease of tongue, *Clin.* 183, 184
- SYMONDS, CHARTERS.—Discussion on excision of larynx and parts of pharynx and œsophagus for carcinoma, *Clin.* 47
- , discussion on pneumococcal colitis with hyperpyrexia, severe hæmorrhage treated by appendicostomy, *Clin.* 51
- on rheumatic spondylitis with torticollis and subluxation, *Clin.* 38
- , excavating rodent ulcer of face under treatment by radium, *Clin.* 74
- , removal of frontal bone for septic osteomyelitis, *Laryng.* 40
- , rodent ulcer treated by radium, *Clin.* 162
- , seconds vote of condolence on death of Mr. H. H. Clutton, *Clin.* 30
- Symphysiotomy, merits of, *Obst.* 116
- , method of delivery in third degree of contracted pelvis, *Obst.* 115
- , points in technique, *Obst.* 118
- , subcutaneous, *Obst.* 122
- Synapse or receptive substance, in relation to contact of neurones, *Path.* 28
- Syncope (cardiac), following myocarditis, cause of sudden death in purulent pericarditis, *Med.* 66
- Syphilide, small follicular, of corymbose type (H. G. Adamson), *Derm.* 1
- Syphilis, cases of, illustrating importance of Wassermann reaction (J. E. R. McDonagh), *Derm.* 90
- complicating pregnancy, treatment, *Surg.* 237, 238
- , decrease in virulence, *Surg.* 209, 210
- , destructive ulceration of hard and soft palate apparently of specific origin in tuberculous subject (J. Dundas Grant), *Laryng.* 71
- , diagnosis of, *Surg.* 210
- by complement-fixing test, *Surg.* 227, 239
- by examination of cerebrospinal fluid, *Surg.* 234
- by presence of *Spirochæta pallida*, *Surg.* 219, 242
- by serum-test, *Surg.* 210
- , Hecht's reaction, *Surg.* 220, 221, 237, 228, 239
- , Wassermann's reaction, *Derm.* 40, *Surg.* 219, 220, 227, 229, 231, 232, 238

- Syphilis, diagnosis of by serum-test, Wassermann's reaction, certainty when test positive, even in absence of signs and symptoms, *Neur.* 120
- , diseases of nervous system due to (F. W. Mott), *Neur.* 35; (H. Head), *Neur.* 49; discussion (p. 60): Dr. Gordon Holmes, Dr. Wilfred Harris, Dr. Grainger Stewart, Dr. Stanley Barnes
- , essential cause of tabes dorsalis and general paralysis, *Med.* 43
- , history of, in case of mycosis fungoides, *Derm.* 40
- , hypertrophic laryngitis with stenosis in subject of (W. Hill), *Laryng.* 93
- , and idiocy, causal relation between, discussed, *Neur.* 120, 122
- , infection between apes, *Neur.* 44
- , —, cause of variation in effects following, *Med.* 44, 45
- , immunity to, for life, conferred by one attack, *Neur.* 48
- , —, —, experiments to prove, *Neur.* 48
- , instruction respecting improvements suggested, *Surg.* 225, 237, 247
- , mixed infection in, treatment, *Surg.* 237
- , in mother and child, treatment, *Surg.* 231, 232
- , of nervous system, cases of, within six years of infection (H. Head), *Neur.* 49
- , —, classification of forms, *Neur.* 35
- , —, concomitant manifestations, *Neur.* 60
- , —, investigation of suspected case, *Neur.* 38
- , —, prodromal symptoms, *Neur.* 50
- , —, treatment, *Neur.* 60, 63
- , —, Wassermann's reaction negative in, *Neur.* 61
- , pathology, advances in, *Surg.* 241
- , prophylaxis, *Surg.* 228, 242
- , —, in British Army in India, *Surg.* 226
- , —, by state control of prostitution, *Surg.* 228, 247
- , ratio to other venereal diseases, constant, *Surg.* 209
- , in relation to kraurosis vulvæ, *Obst.* 38
- , —, to leucoplakic vulvitis, *Obst.* 39
- , treatment by arsenic and aryl-arsenates, *Surg.* 216, 225, 231
- , —, of, discussion on, opened by Mr. J. Ernest Lane, continued by Dr. Alexander Fleming, Major H. C. French, R.A.M.C., Captain L. W. Harrison, Mr. J. E. R. McDonagh, Mr. C. F. Marshall, Dr. George Pernet, Mr. H. W. Bayly, Mr. Jonathan Hutchinson, Mr. J. Ernest Lane (reply), *Surg.* 209-248
- , —, by excision of chancre, *Surg.* 229, 234
- , —, by iodides, *Surg.* 216, 225, 245
- , —, intermittent as opposed to continuous, *Surg.* 230
- , —, by mercury, *Surg.* 211, 222
- , —, duration, *Surg.* 215, 223, 246
- , —, preparations in intramuscular injections, *Surg.* 212, 214, 233, 235, 245, 247, 248
- , —, —, inunctions, *Surg.* 214, 226
- , —, —, oral administration (pills), *Surg.* 230, 234, 235, 240, 243
- , —, —, should be prolonged, *Surg.* 215, 236
- , —, —, should be in physician's as much as surgeon's hands, *Surg.* 242
- , —, by quinine and salicin, *Surg.* 237
- , —, by vaccine, *Surg.* 229
- , —, by serum injections, failure of, *Surg.* 229
- , —, time to commence, *Surg.* 210, 222, 227, 242
- , vascular lesions of brain due to, effects in two cases, *Neur.* 30, 31
- , virus of, aptitude for attacking nervous system, *Neur.* 43
- , —, —, illustrative cases, *Neur.* 43, 44
- , —, attacking or sparing ova, *Neur.* 46
- , —, immunization effected by agency of nerve cells, *Neur.* 47

- Syphilis, see also *Erythema nodosum*, *syphilitic*; *Onychia sicca syphilitica*; *Phlebitis, nodular, syphilitic*
- Syphilis (cerebral), classification of forms, Neur. 35, 36
- , clinical manifestations, Neur. 50
- , exaltation in, Neur. 51
- (cerebrospinal), combination of symptoms pointing to, Neur. 59
- (congenital), case of (L. G. Parsons), Child. 138
- , causing optic atrophy and ultimately leading to dementia paralytica juvenilis (H. Head), Neur. 33
- , Colles's law in, Neur. 45
- , in subjects of idiocy, Neur. 119, 120, 122
- , irregularity of appearance in children of same family, Neur. 46
- , law of Profeta in, Neur. 45
- , parasyphilis following on later, Neur. 46
- , sex-incidence equal, Neur. 43
- , treatment, Surg. 231, 232, 243
- (quaternary), reasons against theory of, Neur. 47
- (secondary), acute meningitis during, Neur. 37
- , lymphocytosis of cerebrospinal fluid in, Med. 36, 37
- , probable late secondary specific pharyngitis with nerve symptoms (J. Dundas Grant), Laryng. 154
- , in relation to arthritis, Baln. 92
- , see *Pharyngitis, secondary, specific*
- (spinal), classification of forms, Neur. 35, 36
- , clinical manifestations, Neur. 55
- , disseminated, diagnosis from disseminated sclerosis, Neur. 63, 64
- , lesions in, Neur. 58
- , latent, Neur. 37, 38
- , post-mortem appearances, Neur. 60, 61
- , root-lesions in, Neur. 57
- (tertiary), elephantiasis associated with (H. G. Adamson), Derm. 81
- (gummatous teno-synovitis) (J. H. Sequeira), Derm. 20
- Syphilitic infection, diagnosis incomplete, R.S.M. 20
- , meningitis, chronic, with compression of cord (T. Grainger Stewart), Neur. 33
- , with compression paraplegia (T. Grainger Stewart), Neur. 20
- , relation of syphilitic eruption of skin to, Neur. 36
- , with universal arteritis, Neur. 51
- , nodule in brain, cause of olfactory discharges, Neur. 3
- , poliomyelitis (W. Harris), Neur. 13
- (tertiary), infiltration of larynx (Dan McKenzie), Laryng. 70
- , ulcerations always complicated by secondary infections, R.S.M. 20
- , of lower lip, inside of cheek and tongue (C. Nourse), Laryng. 68
- , of nasal septum, Neur. 51
- Syphilistics, care of teeth in, Surg. 236
- , children of, results of examination under serum reaction, Neur. 120
- , serum of, complement-fixing power, Surg. 239
- Syphilization, racial, effects of, Neur. 45
- , illustrated by natives of Uganda, Neur. 15
- Syringo-cystadenoma and tricho-epithelioma papulosum, case of mixed tumour (J. E. R. McDonagh), Derm. 32
- Syringomyelia (Morvan's type) in child, shown by symmetrical trophic lesions of extremities (H. Morley Fletcher), Clin. 98
- , rarity of all forms of, among children, Clin. 103
- , with Brown-Séquard syndrome (H. R. Prentice), Neur. 100
- , with total anaesthesia (W. Harris), Neur. 101

- Tabes, with amyotrophy (Cecil Wall), *Neur.* 27
 —, see also *Deafness, tabetic*
 ? Tabes dorsalis, affection of posterior roots of lower cervical and upper dorsal nerves? ; lateral sclerosis (H. Campbell), *Neur.* 34
 Tabes dorsalis, aetiology identical with that of general paralysis, *Neur.* 42
 —, in case of tremor, *Neur.* 19
 —, diagnosis from pseudo-tabes, *Neur.* 39
 —, by Wassermann's reaction, *Neur.* 40-42, 61
 —, increase of proteid in cerebrospinal fluid in, *Neur.* 39
 —, infection of two or more individuals with, from same syphilitic source, *Neur.* 43, 44
 —, lymphocytosis of cerebrospinal fluid in, *Neur.* 41, 42
 —, physical sign common to general paralysis and, *Neur.* 43
 —, small percentage of syphilitic individuals attacked with, *Neur.* 43
 —, sex-incidence equal, *Neur.* 43
 —, treatment with mercury, *Neur.* 42
 —, uselessness and injuriousness of antisyphilitic remedies in, *Neur.* 49
 Tadpoles, experiments on, to test truth of neurone theory, *Path.* 6-8
 Talbot, on absorption of roots of permanent teeth, quoted, *Odont.* 15
 Talipes, prevention, by use of continuous current, *Electr.* 105
 Tambour, use of, in auscultation, advantages over stethoscope, *Med.* 209
 Tampons, use of in subinvolution of uterus, with displacement, *Obst.* 227, 228
 Tapping and incision in treatment of empyema, *Clin.* 121
 —, of pyæmic knee- and shoulder-joints (E. M. Corner and M. Huggins), *Clin.* 120
 TARGETT, J. H.—Adenofibromatous polypus of uterus, showing early malignant disease, *Obst.* 155; discussion (p. 157): Dr. Macnaughton-Jones
 —, carcinoma of the body of the uterus, with keratinization of the growth, *Obst.* 153
 —, discussion on squamous-celled carcinoma of a dermoid cyst of the ovary, *Obst.* 145
 Tarsectomy (partial) for tuberculous disease of foot (C. P. Childe), *Child.* 158
 TATE, W. W. H.—Discussion on abdominal hysterectomy for necrosed and suppurating sub-peritoneal fibroid, *Obst.* 66
 —, myxofibroma of an ovary removed under spinal anaesthesia, *Obst.* 73
 —, pregnancy in a septate uterus treated by abdominal hysterectomy five months after death of foetus at full term, *Obst.* 190; discussion (p. 192): Dr. Maxwell
 —, primary carcinoma of the Fallopian tube associated with acute inflammatory mischief, *Obst.* 75; discussion (p. 77): Mr. Doran, Dr. Spencer, Dr. Macnaughton-Jones
 TAYLOR, GORDON.—Case of Schlatter's disease, *Clin.* 20
 TAYLOR, JAMES.—Discussion on cases of thrombosis of cerebral arteries and cerebral hemorrhage from luetic vessels, *Neur.* 32
 —, discussion on case of tremor, *Neur.* 19
 —, left hemiplegia and left third nerve paralysis, *Neur.* 18
 —, recurring left-side convulsions, with transient motor aphasia, in a left-handed woman, *Neur.* 17
 Teeth, absorption of, associated with infective disease of the jaws, *Odont.* 9-19
 —, care of, important in treatment of duodenal ulcer, *Surg.* 105
 —, in syphilitics, *Surg.* 236
 —, colour of, in relation to resistance to caries, *Odont.* 80, 82
 —, condition of, in skull of Egyptian mummy, *Odont.* 154, 155
 —, decalcifying action of lactic acid on, *Odont.* 74
 —, in dentigerous cysts, nature of, *Odont.* 159
 —, different, relative susceptibility of, to dental caries, experiments on (Stanley P. Mummery), *Odont.* 71
 —, diseases of, and physical deterioration, *Odont.* 5
 —, friction of, natural and artificial preventive of decay, *Odont.* 82
 —, histology of, *Odont.* 127-132
 —, inflammation at roots of, bacterial origin, *R.S.M.* 15

- Teeth, inflammation at roots of, treatment by vaccine, R.S.M. 15
 — of pig, effect of feeding with madder on, Odont. 80, 83
 —, occlusion of, correct in Australian aboriginals, Odont. 156
 —, —, imperfect, Odont. 156
 —, organic and inorganic components of, Odont. 73
 —, resistant powers of, to action of acid based on natural classification, Odont. 75, 77
 —, susceptibility of, to caries, Obst. 78, 79
 — to decay, inherited, Odont. 79
 — (unerupted), method of ascertaining relative position by film radiographs (C. A. Clark), Odont. 87
- Telangiectases in cases of angioneurosis, Clin. 106
 —, hereditary, multiple, with epistaxis (F. Langmead), Clin. 109, 110
- Telegraph messengers, incidence of measles among, Epid. 49
- Temperate zones, subjects of thyroid-gland insufficiency preponderate in, Baln. 5, 6
- Temperature, effect of administration of tuberculin in minute doses on, R.S.M. 118, 149, 151
 — no guide to nature of pericardial effusion, Med. 97
 —, relation to opsonic index, R.S.M. 33, 41, 42, 123, 211, 212
 —, slight rise of, in puerperal morbidity, Obst. 223
- Temporal bone, endothelioma of, cases of (J. Dundas Grant), Otol. 32; D. R. Paterson, Otol. 61
 —, fracture of, specimen (G. J. Jenkins), Otol. 88
 — (right), malignant growth of, with extension through external meatus, resembling aural polypus (P. Watson Williams), Otol. 85
 —, osteomyelitis of (H. Tod), Otol. 57
- Temporo-sphenoidal abscess, case in which vomiting was entirely absent (A. L. Whitehead), Otol. 73
- Tenesmus in intussusception, Child. 168
- Teno-synovitis, gummatous (J. H. Sequeira), Derm. 20
- Teratoma of pineal gland, Neur. 76
 — (retroperitoneal), connected with spinal canal (R. Johnson and T. W. P. Lawrence), Path. 43
- Testicle, intra-abdominal grafting of tissue from, in fowls, Path. 132, 134, 135
- Test-meals in duodenal ulcer, Surg. 74, 92, 112
- Tests, post-operative, summary of, in eight cases of labyrinthine disease (R. Lake and N. Pike), Otol. 2
- Tetanus, bacilli in, destruction, Med. 40
 —, —, generalization, Med. 40
 —, conservation of strength in, Med. 41
 — mortality, causes of, Med. 40
 — from, at Kasr-el-Ainy Hospital, Cairo (1906-8), Med. 39, 54
 —, —, at St. Bartholomew's Hospital (1895-1907), Med. 39, 54
 —, spasms in, relief of, Med. 41
 —, treatment by antitoxin, curative, Med. 40, 53
 —, —, prophylactic, Med. 40
 — by carbolic-acid solutions injected hypodermically, Med. 40
 —, indications in, Med. 40
 — by magnesium-sulphate solution, combined with antitoxin, Med. 41
 — — — — in intraspinal injections (L. Phillips), Med. 39
 — — — — — — — — — — details and résumé of cases, Med. 43, 46, 51
 — — — — — — — — — — in subcutaneous injections, Med. 50
- Tetany in duodenal ulcer, Surg. 81
- Therapeutics, discoveries in, exploitation of, R.S.M. 108
 —, teaching of, in hospital wards (Sir T. Clifford Allbutt), Therap. 1-16
- Thermometer, clinical accuracy of record, not portability, essential, R.S.M. 77
- Thermo-penetration, R.S.M. 157
- Thibierge, kraurosis vulvae, quoted, Obst. 35

Thigh-bone, see *Femur*

Thirst, insatiable, accompanying case of tumour of pituitary body, *Neur.* 72

Thompson, pathology of acne, quoted, *Path.* 173

THOMPSON, A. RALPH.—Case of extracapsular fracture of neck of thigh-bone in boy aged 6,

Child. 22; discussion (p. 22): Mr. P. Lockhart Mummery

—, discussion on case of prolapse of intestine through ectopia vesicæ, *Child.* 55

— on separation and displacement forwards of lower epiphysis of femur, *Child.* 57

THOMPSON, H. THEODORE.—Cervical ribs, *Clin.* 155; discussion (p. 156): Dr. Parkes Weber,

Dr. Thompson (reply)

—, discussion on bluish pigmentation of skin, *Clin.* 142

—, extensive myositis ossificans, with pathological report by Hubert Turnbull, *Clin.* 151;

discussion (p. 154): Dr. E. I. Spriggs, Dr. Parkes Weber, Mr. A. Pearce Gould,

Dr. Thompson (reply)

Thompson, Reginald, insurability of lives the subjects of hydrothorax or empyema in childhood, quoted, *Surg.* 55

Thomson, Professor A., differentiation between male and female pelvis, quoted, *Path.* 110

—, on epiphysial line, quoted, *Child.* 57

THOMSON, GEORGE.—Case showing the result of extraction of the six-year-old molars, *Odont.*

156; discussion (p. 157): Mr. C. F. Rilot, Mr. Rushton, Mr. Robbins, Mr. Thomson

(reply)

—, discussion on susceptibility of teeth to dental caries, *Odont.* 82

THOMSON, H. CAMPBELL.—Case of pontine thrombosis causing anæsthesia of the fifth nerve,

and hemianæsthesia of the same side, *Neur.* 79; discussion (p. 85): Dr. Hinds Howell,

Dr. R. F. Kennedy, Sir Victor Horsley, Dr. Gordon Holmes, Mr. Sydney Scott, Dr.

Purves Stewart, Dr. Henry Head, Dr. Campbell Thomson (reply), Dr. Wilfred Harris

(reply)

THOMSON, STCLAIR.—Complete inspiratory stenosis of the larynx in a man, case for

diagnosis, *Laryng.* 112; discussion (p. 113): Dr. Paterson, Dr. Milligan, Dr. F. Powell,

Dr. StC. Thomson (reply)

—, complete stenosis of larynx left after diphtheria and tracheotomy in a boy when aged

1 year and 2 months, completely cured by repeated intubations spread over three

years, *Laryng.* 35; discussion (p. 36): Mr. Herbert Tilley, Dr. StC. Thomson (reply)

—, discussion on bands between Eustachian tube and pharynx, *Laryng.* 70

— on case of laryngeal new growth for diagnosis, *Laryng.* 62

— of pemphigus vegetans affecting larynx, &c., *Laryng.* 78

— of tuberculosis of pharynx, *Laryng.* 59

— on endothelioma of ethmoid, *Laryng.* 67

— on extrinsic carcinoma of larynx, *Laryng.* 54

— on Kuhn's instruments for per-oral intubation, *Otol.* 49

— on large nasopharyngeal growth in a boy aged 12, *Otol.* 48

— on papillomata of larynx from a boy aged 6 removed by direct method, *Laryng.* 98

— on removal of a rapidly-growing soft fibroma from posterior wall of left maxillary

antrum by a modified Rouge's operation, *Laryng.* 96

— on "singer's node" on left cord, *Laryng.* 108

— on specimen of larynx showing redevelopment of epithelioma on left cord after

removal of right for same cause, September, 1896, *Laryng.* 34

— on vaccine therapy, *R.S.M.* 51

—, extensive tuberculosis of epiglottis, left ary-epiglottic fold, and left inter-arytænoid space,

in a gentleman aged 47, completely healed by the galvano-cautery and sanatorium

treatment, *Laryng.* 7; discussion (p. 8): Mr. Tilley, Dr. Watson Williams, Dr. Scanes-

Spicer, Dr. Donelan, Dr. Dundas Grant, Dr. StC. Thomson (reply)

—, four cases of epithelioma of the larynx after operation, *Clin.* 115; discussion (p. 118):

Mr. A. E. Barker, Dr. Heron, Mr. Burghard, Mr. Harmer, Dr. StC. Thomson (reply)

—, papilloma of the larynx in a boy aged 6½ of four years' duration, cured by tracheotomy

and repeated operations by direct laryngoscopy, *Laryng.* 11; discussion (p. 12): Dr.

Paterson, Dr. StC. Thomson (reply)

- THOMSON, STCLAIR.—Tuberculosis of both vocal processes in a medical man aged 41, cicatrized with seven applications of the galvano-cautery, *Laryng.* 6; discussion (p. 8): Mr. H. Tilley, Dr. Watson Williams, Dr. Spicer, Dr. Donelan, Dr. Dundas Grant, Dr. StC. Thomson (reply)
- , tuberculosis of all the left vocal cord and inter-arytenoid space in a lady aged 46, completely healed by two months' silence and sanatorium treatment, *Laryng.* 6; discussion (p. 8): Mr. H. Tilley, Dr. Watson Williams, Dr. Scanes Spicer, Dr. Donelan, Dr. Dundas Grant, Dr. Thomson (reply)
- , two cases of radical frontal sinus operation, to show the conditions nine and sixteen months after operation, *Laryng.* 144; discussion (p. 144): Dr. Watson Williams, Dr. J. Dundas Grant, Dr. Thomson (reply)
- , unilateral pansinusitis in a man aged 21, shown six weeks after operation, when all four cavities were operated on at one sitting, *Laryng.* 114
- , woman aged 56, after laryngo-fissure for subglottic enchondroma, *Laryng.* 13; discussion (p. 13): Mr. S. Green, Dr. Thomson (reply)
- THOMSON, THEODORE, C.M.G.—Discussion on measles and its pre-eruptive symptoms, *Epid.* 66
- Thoracoplasty in treatment of very large empyema in two operations (J. E. Adams), *Clin.* 90
- Thorax, congenital deformity of (E. I. Spriggs), *Child.* 102
- , dorsal percussion of (W. Ewart), *Med.* 211
- , see also *Aneurysm* (thoracic)
- THORNE, ATWOOD.—Case for diagnosis, *Laryng.* 92; discussion (p. 92): Dr. Dundas Grant, Mr. Atwood Thorne (reply)
- , discussion on case for diagnosis, *Laryng.* 140
- , — of probable malignant disease of larynx in woman, *Laryng.* 37
- THRESH, J. C.—Discussion on scarlet fever, home treatment and prevention, *Epid.* 71
- Throat, lupus of, treatment by tuberculin inoculations, *R.S.M.* 51-52
- , painful, case of, treatment by pneumococcus vaccine, *R.S.M.* 184
- and larynx, variation in effect of "back" and "belly" breathing on stresses, strains, and frictions in, illustrated by model (R. H. Scanes Spicer), *Laryng.* 41
- and nose, lupus of, treatment discussed, *Laryng.* 87
- Throb, production of, under simultaneous employment of stethoscope with armlet, *Med.* 208, 209
- Thrombosis of cerebral arteries, syphilitic (H. Head), *Neur.* 30
- of left posterior cerebellar artery followed by severe trigeminal neuralgia in analgesic facial area (W. Harris), *Neur.* 81
- (pontine), case of, causing anaesthesia of fifth nerve and hemianæsthesia of same side (H. Campbell Thomson), *Neur.* 79
- (venous), acute cerebral softening possibly due to (W. Harris and B. H. Spilsbury), *Neur.* 130
- and necrobiosis of tumour, with fibromyoma and pregnancy (T. G. Stevens), *Obst.* 54
- THURSFIELD, HUGH.—Discussion on case of compression of trachea by enlarged thymus, *Child.* 48
- THURSTON, E. O.—Chylous cyst excised from the neck, *Path.* 97
- Thymus gland as direct cause of death, *Child.* 47, 48
- , enlarged, causing compression of trachea (G. Carpenter), *Child.* 46
- , condition of, and lymphatism, *Anæsth.* 51
- , lymphatism associated with, cases, *Anæsth.* 21-30
- , see also *Lymphatism*
- , grafting of, in animals, experiments on, *Path.* 135
- , persistent, physical sign of lymphatism, *Anæsth.* 52
- secretion, possible relation to sudden death under chloroform, *Anæsth.* 51
- Thyroid cartilage, perichondritis of, of unknown origin (H. Barwell), *Laryng.* 109
- , transverse axis of respiratory rotation of cricoid on, in belly-breathing, illustrated by model (R. H. Scanes Spicer), *Laryng.* 41, 44, 49

- Thyroid gland, diet in relation to, Baln. 10
- — —, engorgement of, cardiac and respiratory failure under anaesthetic, associated with, *Anæsth.* 45
 - — —, enlargement produced by ingestion of radio-active waters, Baln. 9
 - — —, symmetrical, physical sign of lymphatism, *Anæsth.* 52
 - — —, extract, administration to dachshund, with negative result, *Path.* 108
 - — —, previous administration in cases of lymphatism, *Anæsth.* 58
 - — —, in treatment of adiposis dolorosa, *Clin.* 59, 60
 - — —, functional activity, diuresis sign of, Baln. 9
 - — —, essential to pregnancy, Baln. 10
 - — —, stimulated by calcium salts, Baln. 9
 - — —, insufficiency and lymphatism, *Anæsth.* 43
 - — —, gouty and rheumatoid arthritis due to, Baln. 8
 - — —, group of diseases probably due to, amenability to balneary therapeutics, Baln. 8, 9
 - — —, metabolic osteo-arthritis due to, Baln. 8
 - — —, subjects of, preponderate in temperate zones, Baln. 5, 6
 - — —, internal secretion of, promotion of normal growth of bones by, *Path.* 138, 139
 - — —, operation on, cases illustrating beneficial effects of, on extra-laryngeal inoperable carcinoma (W. Stuart-Low), *Laryng.* 13
 - — —, part of, gullet, larynx, and four rings of trachea removed during act of suicide (E. A. Peters), *Laryng.* 107
 - — —, larynx, four rings of trachea, and gullet removed during an act of suicide (E. A. Peters), *Larynx.* 107
 - — —, partial removal of, followed by laryngeal paralysis (S. Hastings), *Laryng.* 150
 - — —, preparations, action of (W. Edmunds), *Path.* 100
 - — —, secretion diminished, effect on bone growth, *Path.* 106
 - — —, deficiency, effect on limbs of animals, *Path.* 107
 - — —, effect on condition of blood-plasma, Baln. 7, 8
 - — —, functions of, Baln. 8
- Thyrotomy for epithelioma of left vocal cord (J. Dundas Grant and D. McKenzie), *Laryng.* 19
- — —, of right vocal cord (J. Dundas Grant), *Laryng.* 17
- Tibia, enlargement of tubercle of (Schlatter's disease) (G. Taylor), *Clin.* 20; (R. C. Elmalie), *Clin.* 189
- — —, see also *Schlatter's disease*
- Tibio-calcaneal resection at ankle, with amputation of foot, case of (C. Gordon Watson), *Clin.* 133
- Tick-bites in horse, eosinophile cells exudate from (J. B. Cleland), *Path.* 42
- Tics (clicking), prognosis bad, *Clin.* 94, 95
- TIDY, H. L., WOODS, R. S., and ZUNDEL, C. E.—Statistics of pericardial effusion from the London Hospital, *Med.* 115
- Tileston, study of weight in measles, quoted, *Epid.* 52
- TILLEY, HERBERT.—Case of audible tinnitus, *Otol.* 1; discussion (p. 10): Mr. Baber, Dr. H. J. Davis, Mr. Waggett, Mr. C. G. Lee, Dr. FitzGerald Powell, Dr. Law, Mr. A. L. Whitehead, Mr. Tilley (reply)
- — —, case of healed tuberculous laryngitis, treated by galvano-puncture, *Laryng.* 121; discussion (p. 121): Dr. Jobson Horne, Dr. D. McKenzie, Dr. Scanes Spicer, Dr. Donelan, Dr. F. Powell, Mr. Horsford, Dr. W. Hill, Dr. J. Dundas Grant, Mr. H. Tilley (reply)
 - — —, discussion on atresia of the anterior naris, *Laryng.* 116
 - — —, on case of chronic laryngitis, with ? prolapse of ventricle on right side, *Laryng.* 30
 - — —, of complete stenosis of larynx following diphtheria and tracheotomy, cured by repeated intubations, *Laryng.* 36
 - — —, of large chronic swelling in tonsillar region, *Laryng.* 133
 - — —, of laryngeal new growth for diagnosis, *Laryng.* 61
 - — —, of laryngeal paralysis, *Laryng.* 150

- TILLEY, HERBERT.—Discussion on chronic glanders in a man, *Laryng.* 2
 —, discussion on early symptoms in case of epithelioma originating near right Eustachian tube, *Otol.* 29, 31
 — on endothelioma of ethmoid, *Laryng.* 67
 — on extrinsic carcinoma of larynx, *Laryng.* 54
 — on foreign bodies in lung and œsophagus, *Laryng.* 77
 — on growth in nasopharynx resembling pale adenoids, *Laryng.* 73
 — on question of operation for complete occlusion of left nostril by deflected triangular cartilage in a child, *Laryng.* 58
 — on removal of hypertrophied anterior lip of hiatus semilunaris, *Laryng.* 132
 — on sanatorium, galvano-cautery and silence in treatment of tuberculosis of throat, *Laryng.* 8
 — on "singer's node" on left cord, *Laryng.* 107
 — on treatment of stricture of rectum and other conditions by fibrolysin, *Clin.* 89
 — on two cases of hoarseness, *Laryng.* 41
 —, objective clicking sound in left ear, *Otol.* 36; discussion (p. 37): Dr. Law, Dr. Paterson, Dr. Grant, Dr. McBride, Mr. Tilley (reply)
 — and rhythmical "clicking" noise in left ear, associated with synchronous muscular contraction of certain muscles forming the floor of the mouth, *Clin.* 93; discussion (p. 94): Mr. Tilley, Dr. Morley Fletcher, Dr. Henry Head, Mr. Tilley (reply)
 —, papillomata of larynx from boy, aged 6, removed by direct method, *Laryng.* 97, 124; discussion (pp. 97-99): Dr. Watson Williams, Dr. Jobson Horne, Dr. StClair Thomson, Mr. Mark Hovell, Dr. F. Powell, Dr. J. Dundas Grant, Mr. Waggett, Dr. Paterson, Mr. Tilley (reply); (p. 124): Dr. J. Dundas Grant, Mr. Tilley (reply)
 —, penny removed from the gullet of a young girl, *Laryng.* 135; discussion (p. 135): Dr. S. Spicer, Dr. Dundas Grant, Dr. E. Davis, Mr. Tilley (reply)
 —, portion of mutton bone removed by the direct method from right bronchus of lady, in whom it had been lodged for ten days, *Laryng.* 127
 —, removal of a rapidly-growing soft fibroma from posterior wall of left maxillary antrum by a modified Rouge's operation, *Laryng.* 96; discussion (pp. 96-97), Dr. Watson Williams, Mr. Rose, Dr. StClair Thomson, Dr. Dundas Grant, Mr. C. Nourse, Mr. Tilley (reply)
 —, tuberculosis of epiglottis treated by amputation of diseased portion, *Laryng.* 32; discussion (p. 32): Mr. Harold Barwell
 —, two cases of suppurating dental cysts invading the maxillary antrum, *Odont.* 115; discussion (p. 117): Mr. C. Schelling, Mr. J. F. Colyer, Mr. W. Hern, Mr. Tilley, (reply)
 —, vulsellum-catch forceps for firmly securing submerged tonsils in the operation for their more efficient removal, *Laryng.* 124; discussion (p. 124): Dr. Jobson Horne, Dr. Donelan, Dr. F. Powell, Mr. C. Fox, Mr. Stuart-Low, Dr. Scanes Spicer, Dr. W. Hill, Dr. D. McKenzie, Dr. Peters, Dr. J. Dundas Grant, Mr. Tilley (reply)
 —, when is cancer cured? Larynx of a man, aged 76, whose right vocal cord was removed for epithelioma in September, 1896, *Laryng.* 33; discussion (p. 34): Mr. Tilley, Dr. Dundas Grant, Dr. H. J. Davis, Mr. de Santi, Dr. StClair Thomson, Dr. Middlemass Hunt, Dr. Watson Williams, Dr. Jobson Horne
 Tinnitus, audible, case of (A. Sharp), *Otol.* 54
 — — — (H. Tilley), *Otol.* 1
 THARD, NESTOR.—Discussion on oxaluria and calcium-oxalate deposit, *Med.* 23
 Tissue-degeneration, toxic products of, *R.S.M.* 85
 Tissue-inoculation, homologous or autologous, equally protective against cancer, *Therap.* 76, 77
 Titus, Dr., explanation of static wave current, quoted, *Electr.* 106
 Tlascalans, abstinence from use of salt, by, *Baln.* 60
 Tobacco as cause of hypersecretion, *Surg.* 181
 Tobacconist, intermittent claudication in, due to chronic arteritis obliterans, *Clin.* 96

- Tod, H.—Case in which the clinical symptoms simulated a cerebellar abscess; brain explored on two occasions; no abscess discovered; recovery, *Otol.* 79; discussion (p. 80): Dr. D. McKenzie
- , discussion on adventitious membrane, resembling normal drum, formed after radical mastoid operation, *Otol.* 59
- on deformity of both pinnae, *Otol.* 72
- exostosis of right external meatus in a boy aged 10, *Otol.* 76; discussion (p. 76): Mr. H. Mole, Mr. West, Mr. Whitehead, Dr. E. Law, Dr. Pritchard, Dr. McKenzie, Dr. Milligan, Dr. Westmacott, Mr. Cheate, Mr. Tod (reply)
- , osteomyelitis of the right temporal bone, secondary to mastoid disease; removal; recovery, *Otol.* 57; discussion (p. 58): Dr. H. J. Davis, Mr. Tod (reply)
- , specimen of cholesteatoma or keratosis obturans of the external auditory canal, *Otol.* 81
- Toe-nails, see *Finger- and toe-nails*
- Tongue, cheek, lips, larynx, fauces and nasal cavities affected by pemphigus vegetans (H. J. Davis), *Laryng.* 78
- , entire excision of, with results, case of (H. J. Davis), *Laryng.* 137
- , epithelioma of, without keratinization (F. S. Eve), *Clin.* 180
- , lower lip and inside of cheek, syphilitic ulceration of (C. Nourse), *Laryng.* 68
- , lymphangioma of, cases (E. Rock Carling, A. E. Barker), *Clin.* 111, 112
- , treatment, operative, *Clin.* 112
- , malignant disease of, diagnosis of various forms, *Clin.* 186
- , surgery (F. S. Eve), *Clin.* 173
- , papille of, prominence, physical sign of lymphatism, *Anæsth.* 52
- , spasm of, accompanying myoclonus (W. Harris), *Neur.* 104
- , surgery of, conservative, *Clin.* 186, 187
- , tuberculous ulcer (large) on right side of (H. J. Davis), *Laryng.* 81
- , tumours of base of (three rare, two of them endotheliomata) (F. S. Eve), *Clin.* 173
- Tonsil (lingual), hypertrophy of, with impairment of singing voice, improved by snaring operation (J. Dundas Grant), *Laryng.* 155
- , lymphosarcoma, removed by latent pharyngotomy after Vohsen's method (G. Wilkinson), *Laryng.* 86
- , neoplasm of (G. S. Hett), *Laryng.* 2
- , new instrument for operations on, *Laryng.* 134
- (right), new growth of, invading surrounding tissues (A. Evans), *Laryng.* 147
- , tuberculosis of, associated with tuberculous glands of neck (E. Scott Carmichael), *Child.* 27-36
- , microscopical appearances, *Child.* 34
- Tonsils, exudation from, general sign of impure scarlatinal infection, *Epid.* 80
- , hyperplasia, physical sign of lymphatism, *Anæsth.* 52
- , large chronic swelling in tonsillar region, with enlarged gland in neck: ? sarcoma (W. Hill), *Laryng.* 132
- (submerged), vulsellum-catch forceps for securing (H. Tilley), *Laryng.* 124
- , tuberculosis of, microscopical appearances insignificant, *Child.* 33
- , primary, *Child.* 29, 30
- , secondary, *Child.* 28, 29, 30
- Toothache, bacterial origin, *R.S.M.* 15
- , treatment by vaccine, *R.S.M.* 15
- Tooth-follicle in herbivora, *Odont.* 162
- Tooth, H. H., C.M.G., transmission of typhoid fever by flies, quoted, *Epid.* 214
- Tophi (uratic), deposits of, in cases of gout in boy aged 14 (Sir D. Duckworth), *Clin.* 23
- in Mahommedan (Sir D. Duckworth), *Clin.* 21
- Torsion of body of uterus, specimen of (W. S. A. Griffith), *Obst.* 187
- Torticollis and subluxation, with rheumatic spondylitis (T. H. Openshaw), *Clin.* 37, 38
- Town-life, ill-effects on children of poorest classes, *Epid.* 5
- Towns, casual labourers in, uncertain conditions of livelihood, *Epid.* 6, 8

- Towns, poorest classes in, liability to disease, *Epid.* 4, 5
- Toxemia, cause of death in tetanus, *Med.* 40
- from intestinal absorption, complicating idiopathic dilatation of colon, *Child.* 180
- (general) in lymphatism, *Anæsth.* 57
- Toxins and anti-toxins carry opposite electrical charges, *R.S.M.* 156
- , destruction of cancer-cells by, *Surg.* 11
- (bacterial), in treatment of inoperable sarcoma (W. B. Coley), *Surg.* 1-44
- — — — —, history of method, *Surg.* 2
- (mixed), action on inoperable sarcoma, *Surg.* 9-12
- — — — —, reason why cure results, *Surg.* 11
- — — — —, dangers of, *Surg.* 22
- — — — —, dosage, *Surg.* 20
- — — — —, indications for use of, *Surg.* 12
- — — — —, obstacles to fair trial of, *Surg.* 21
- — — — —, prophylactic against operations for carcinoma, *Surg.* 16
- — — — — recurrence after operation for, *Surg.* 15
- — — — — render tissues unfavourable for sarcoma growth, *Surg.* 12
- — — — — in treatment of malignant disease, *Therap.* 79
- — — — — of sarcoma, duration, *Surg.* 18
- — — — — of inoperable sarcoma, final results, with records and tables of cases, *Surg.* 24-42
- — — — — in sarcoma of long bones, *Surg.* 12-16
- Trachea, compression of, by enlarged thymus (G. Carpenter), *Child.* 46
- , diphtheritic membrane found in, after death, in case of boy who died apparently from uræmia (J. Walter Carr), *Child.* 90
- , four rings of, larynx and part of thyroid gland removed during act of suicide (E. A. Peters), *Laryng.* 107
- and larynx, portions of, with specimen of goitre, from woman (J. Donelan), *Laryng.* 38
- Tracheotomy and diphtheria, complete stenosis of larynx after, completely cured by repeated intubations spread over three years (StC. Thomson), *Laryng.* 35
- and operations by direct laryngoscopy, cause of cure of papilloma of larynx of four years' duration in child (StC. Thomson), *Laryng.* 11
- Tracy, Martha, preparation of mixed toxins of *Bacillus prodigiosus* and *Streptococcus erysipelas*, quoted, *Surg.* 7
- Trade depression, recurring cycles of, cause of pauperism, *Epid.* 10
- — — — —, effect on casual labour, *Epid.* 6, 10, 37
- — — — —, means suggested for provision against, *Epid.* 37
- Transformers, see *High-tension transformer*
- Traube's area, *Med.* 231, 235, 236
- — — — —, increase in size, *Med.* 235
- Traumatism, causing organic disease, with special sense discharges, *Neur.* 12, 15, 16
- , purulent pericardial effusion associated with, *Med.* 125
- , see also *Injuries*
- Tremor, case of (E. Farquhar Buzzard), *Neur.* 18
- , accompanying cases of tumour of pituitary body, *Neur.* 73, 74
- — — — — tumour of pineal body, *Med.* 74, 78
- Trendelenberg position, danger of, *Obst.* 9
- — — — —, importance of, in exposing bladder growths, *Clin.* 72
- Trophining, for general epilepsy, case shown after interval of three years (W. G. Spencer), *Clin.* 65
- Treponema pallidum*, demonstration by ultra-microscope (H. W. Bayly), *Clin.* 3
- — — — —, examination for, in syphilitic lesion, technique, *Clin.* 5
- — — — —, points of distinction, *Clin.* 4
- — — — — from species of *spirochæta*, *Clin.* 4
- — — — —, see also *Spirochæta pallida*

- TREVELYAN, E. F.—Discussion on case of acute cerebral softening, *Neur.* 132
- TREYOR, R. SALUSBURY.—Pathological report on case of lymphatism related by Mr. H. Bellamy Gardner, *Anæsth.* 27
- Triangular cartilage, deflected, complete occlusion of left nostril by, in child (G. Cathcart), *Laryng.* 56
- , operation by removal of projecting cartilage.
- , *Laryng.* 57, 58
- , extreme deflection of, treated by septal fissure (L. H. Pegler), *Laryng.* 128
- Tricho-epithelioma papulosum and syringo-cystadenoma, case of mixed tumour (J. E. R. McDonagh), *Derm.* 32
- Tricuspid valve, malignant endocarditis of, in child aged 6 (F. H. Hawkins), *Child.* 139
- Trilactine, solid preparation containing lactic-acid bacillus, *Therap.* 53, 58, 62
- Tripter, advocacy of electrical treatment in gynæcology, *Obst.* 27
- Trochanters, absence of, in reptilia, development in man, *Clin.* 192
- Trophic lesions (symmetrical) of extremities in child; syringomyelia (Morvan's type) (H. Morley Fletcher), *Clin.* 98
- Tropics (the), marked development of dermic pigmentation amongst whites adapting themselves to, *Baln.* 5
- TROTTER, WILFRID.—Discussion on a case of extrinsic carcinoma of the larynx, *Laryng.* 53
- and POYNTON, F. J.—Case of cardiolysis, *Clin.* 199
- Trypanosomiasis, treatment by arylarsonates, justifiable and imperative, *Surg.* 219
- Tubercle of œcum and hypersecretion, *Surg.* 185
- endotoxin, injections of, effect on opsonizing action of serum of healthy rabbits (R. T. Hewlett), *Path.* 165
- Tuberculides (chronic), case of (J. H. Stowers), *Derm.* 54
- (non-ulcerative) following lupus vulgaris (J. H. Sequeira), *Derm.* 52
- Tuberculin, administration, oral, R.S.M. 118, 152
- , discovery of, exploitation of, R.S.M. 108
- , effect of injection of, and of tubercle endotoxin on opsonizing action of serum of healthy rabbits (R. Tanner Hewlett), *Path.* 165
- not employed in case of pulmonary tuberculosis with other regions involved, R.S.M. 72, 73
- , injections of, in treatment of lupus (G. T. Western), *Derm.* 8
- , ——— of scrofuloderma (G. T. Western), *Derm.* 8
- , preparations of, preference in, R.S.M. 105
- , risks of use of, *Therap.* 17
- , treatment with, by aid of opsonic index, R.S.M. 48
- , cases of apparent failure, R.S.M. 49, 50
- in treatment of anæmic, tuberculous children, results good, R.S.M. 116
- of lupus, R.S.M. 105
- of nose and throat, R.S.M. 51-54
- of laryngeal tuberculosis, R.S.M. 63
- of non-pulmonary tuberculosis, results more satisfactory than in pulmonary form, R.S.M. 115, 116
- of pulmonary tuberculosis, R.S.M. 85, 105
- , ———, combined with sanatorium life, R.S.M. 116
- , ———, failure, R.S.M. 108
- , ———, in minute doses, R.S.M. 118, 149, 151, 207, 209
- , ———, effect on temperature, R.S.M. 118, 149, 151
- , ———, limitations, R.S.M. 119
- , ———, oral administration, R.S.M. 198
- of surgical tuberculosis, R.S.M. 105
- of tuberculosis of local and pulmonary distribution, benefit only partial, R.S.M. 63
- of tuberculous peritonitis, R.S.M. 115
- (new), administration of, R.S.M. 95

- Tuberculin (old), diagnostic injections of, R.S.M. 95
- T. R. in treatment of pulmonary tuberculosis, R.S.M. 150
- emulsion in treatment of pulmonary tuberculosis, R.S.M. 158, 159
- Tuberculosis, cases of, failing under vaccine therapy, R.S.M. 49, 50
- , chronic pancreatitis in, Med. 173
- of conjunctiva, treatment by vaccine inoculations, R.S.M. 61
- , destructive ulceration of hard and soft palate apparently of specific origin in subject of (J. Dundas Grant), Laryng. 71
- of epiglottis, left ary-epiglottic fold, and left inter-arytenoid space, healed by galvano-cautery and sanatorium treatment (StC. Thomson), Laryng. 7
- treated by amputation of diseased portion (H. Tilley), Laryng. 32
- , family predisposition to, as exemplified by infection records of Mount Vernon Hospital Staff, Med. 152
- , hospital infection of, exemplified by records of Mount Vernon Hospital (J. E. Squire), Med. 137-156
- , infection of, by ingestion, Child. 27
- — — — — compared with inhalation, Child. 27, 28
- — — — — primary channels of, Child. 27
- or neoplasm? of left ventricular band (J. Dundas Grant), Laryng. 16
- , pericardial effusion associated with, Med. 56, 65
- — — — — purulent, associated with, Med. 125
- , rarity of, in Chiltern district, Baln. 46
- of serous membranes, case of relation of temperature to opsonic index in, R.S.M. 41, 42
- , treatment by tuberculin, oral administration, R.S.M. 198
- of left vocal cord and inter-arytenoid space, healed by two months' silence and sanatorium treatment (StClair Thomson), Laryng. 6
- of both vocal processes, cicatrized with seven applications of galvano-cautery (StClair Thomson), Laryng. 6
- , see also *Children*, tuberculous; *Glands*, cervical, tuberculous
- , see also *Empyema*, tuberculous; *Pericarditis* (tuberculous); *Phthisis*
- (bovine), distribution of tuberculous farms in Manchester milk supply district, Epid. 225
- , influence of age of cows on, Epid. 235, 241
- , preventable, Epid. 225, 242, 243, 255, 256
- (chronic), treatment by vaccines, results, R.S.M. 147
- — — — —, limitations, R.S.M. 148
- (general), normal or high indices in, R.S.M. 192
- of genito-urinary tract, treatment by vaccine, results, R.S.M. 147
- of hip-joint, acute, treatment by tuberculin with aid of opsonic index, R.S.M. 47
- of knee-joint, recovery after treatment by tuberculin, R.S.M. 115
- (generalized), simulated by pneumococcal infection in infant (J. Walter Carr), Child. 14
- (hyperplastic?) of pelvic colon (J. P. Lockhart Mummery), Clin. 201
- , primary, of stomach and duodenum (W. G. Nash), Clin. 40
- (laryngeal) in girl (G. C. Cathcart), Laryng. 127
- , extensive, in middle-aged man, treatment by galvano-cautery, recovery almost complete (J. Dundas Grant), Laryng. 87
- , site of development in, Laryng. 159, 160
- , treatment by inhalations of pure ether and hexamethylene tetramin, Laryng. 39
- — — — — by tuberculin, R.S.M. 63
- — — — — in pulmonary involvement, R.S.M. 63
- , with considerable dysphagia, treated and relieved by congestion hyperæmia (W. Howarth), Laryng. 164
- , with extreme dysphagia, relieved by injection of alcohol into left superior laryngeal nerve (J. Dundas Grant), Laryng. 152
- (non-pulmonary), treatment by tuberculin more satisfactory than in pulmonary cases, R.S.M. 115, 116

- Tuberculosis (pharyngeal), case of (A. Wylie), Laryng. 58
 — (pulmonary), advanced case, treatment by vaccine inoculation, apparent failure, R.S.M. 36
 — —, febrile case illustrating value of opsonic index, R.S.M. 44
 — —, case with other regions involved, treatment without tuberculin, R.S.M. 72, 73
 — —, coincident with infection in other regions, treatment by tuberculin effects local, not general, improvement, R.S.M. 63
 — —, control of vaccine inoculations by opsonic index, R.S.M. 83
 — — — —, dispensed with in advanced cases, R.S.M. 83
 — —, danger of allowing subjects of, to be employed as bakers, Clin. 119
 — —, diagnosis by opsonic index must be complete, R.S.M. 47
 — — — —, vaccine therapy and, R.S.M. 204
 — —, drugs administered by inhalation with ether, Anaesth. 17
 — —, mixed infection, results of treatment, R.S.M. 206
 — —, prognosis by opsonic index valueless, R.S.M. 200-203
 — —, sanatorium cases, treatment by vaccines for mixed infection, R.S.M. 149, 206
 — —, symptoms a guide to progress and treatment of case, R.S.M. 125
 — —, treatment by continuous antiseptic inhalations (D. B. Lees), Therap. 17
 — — — — by ether inhalations without admixture of exhalations from lungs; successful case (R. H. Hodgson), Clin. 213
 — — — — by muscular exercise electrically produced, suggested, R.S.M. 157
 — — — — by sanatorium life combined with tuberculin in minute doses, R.S.M. 149, 150, 151, 207
 — — — — — —, with use of tuberculin, R.S.M. 116
 — — — — — — of tuberculin emulsion, R.S.M. 158, 159
 — — — — — —, value proved, R.S.M. 109, 110
 — — — — — — by tuberculin, R.S.M. 84, 105, 115, 116
 — — — — — —, failure in, R.S.M. 108
 — — — — — —, limitations, R.S.M. 119
 — — (tonsillar), associated with tuberculous glands of neck (E. Scott Carmichael), Child. 27
 — —, macroscopical appearances insignificant, Child. 33
 — —, primary, Child. 29, 30
 — —, secondary, Child. 28, 29, 30
 Tuberculous abscesses, how diagnosed, R.S.M. 95
 — adenomyoma of Fallopian tube, with tuberculous salpingitis, Obst. 240
 — disease of foot, treatment by partial tarsectomy (C. P. Childre), Child. 158
 — infection of lumbar vertebrae, treatment with tuberculin by aid of opsonic index, R.S.M. 48
 — infection, diagnosis incomplete, R.S.M. 20
 — laryngitis, case healed, treated by galvano-puncture (H. Tilley), Laryng. 121
 — origin of case of multiple serositis, treated by cardiolysis (F. J. Poynton and W. Trotter), Clin. 199, 200
 — pyosalpinx, bilateral (H. J. Paterson), Obst. 80
 — salpingitis, with tuberculous adenomyoma of Fallopian tube (J. Inglis Parsons and B. Glendining), Obst. 238
 — ulcer (large) on right side of tongue (H. J. Davis), Laryng. 81
 — — — — mistaken for urethral calculus, Electr. 91, 92
 — — ulceration of chin, treatment by proteus vaccine, R.S.M. 21
 — — — — of epiglottis, apparently healed by frequent use of electro-cautery, Laryng. 156
 — — ulcerations always complicated by secondary infections, R.S.M. 20
 — — whitlow, how diagnosed, R.S.M. 95
 "Tuberculozyne," fraudulent quack remedy, Therap. 98, 99
 Tumour, abdominal, disappearing, case of (P. Sargent), Clin. 85
 — —, cerebellar, case of (W. J. Maloney), Neur. 99
 — —, cystic, of right broad ligament, springing from uterus and apparently developed from Gartner's duct (A. H. N. Lewers), Obst. 67
 — —, malignant, primary, bile-producing, of liver (F. Parkes Weber), Path. 147

- Tumour, mixed (tricho-epithelioma papulosum and syringo-cystadenoma) (J. E. R. McDonagh), Derm. 32
- , presence of, in gastric cancer, Surg. 134
- , in intussusception, Child. 167, 168
- , of uterus, of doubtful nature (J. Barris), Obst. 73
- , see also under name of *organ or region affected*
- Tumour-cell, soil favourable or unfavourable to, Surg. 11
- Tunica vaginalis, construction of, Surg. 167
- , power of resistance of, Surg. 167
- , rupture of, in hydroceles (S. Hastings), Surg. 165
- Turbinate, inferior, bleeding polypus of (S. Hastings), Laryng. 102-106
- TURNBULL, HUBERT.—Pathological report on case of extensive myositis ossificans, Clin. 151
- TURNBULL, H. H.—Discussion on the bacteriology of pyorrhœa alveolaris, Odont. 64
- , and SEWELL, S. V.—Systematic histological examination of the central nervous system of a case of transverse lesion of the spinal cord in the lower cervical region, Path. 61-72
- TURNER, F. M.—Discussion on scarlet fever, home treatment and prevention, Epid. 67
- TURNER, J. G.—Discussion on bacteriology of pyorrhœa alveolaris, Odont. 63
- , discussion on condition of the mouth in chronic disease, Odont. 105
- TURNER, PHILIP D.—Abdominal hysterectomy sixty-three hours after labour for a necrosed and suppurating subperitoneal fibroid, Obst. 61; discussion (p. 65): Dr. Lewers, Mrs. Boyd, Dr. Spencer, Dr. Tate, Dr. Macnaughton-Jones, Dr. Turner (reply)
- TURNER, H. G.—Two cases of myotonia atrophica, Clin. 145; discussion (p. 147): Dr. F. E. Batten
- Turpentine, injection of, in dogs, not the direct cause of pancreatic reaction in urine, Path. 90
- , producing acute and subacute pancreatitis, Path. 80, 81, 89
- TWEEDIE, A. R.—Model of the upper and lower jaws and impression of the roof of the mouth, from a young man aged 19, Laryng. 141; discussion (p. 141): Mr. Barwell, Mr. Tweedie (reply)
- , specimen of the face and mouth of a female infant, showing arrest of development of the right half of the tongue, combined with a cleft of the soft palate and a palato-lingual fold, Laryng. 99; discussion (p. 101): Dr. Paterson, Mr. Tweedie (reply)
- TWEEDY, E. HASTINGS.—Modern methods of delivery in contracted pelvis, Obst. 115; discussion (p. 121): Dr. Champneys, Dr. Herman, Dr. Herbert Spencer, Dr. Eden, Dr. Griffith, Dr. Macnaughton-Jones, Dr. Tweedy (reply)
- , specimen of ovarian pregnancy (report by R. J. Rowlette), Obst. 132
- Twin, achondroplasia in (R. Hutchison), Child. 41
- Twins, "true" and "false," Child. 43
- Tympanic membrane, structure of, Otol. 64
- Tympanic plates, case of; ? congenital defect of both (C. E. West), Otol. 61
- Typhoid fever, chronic pancreatitis following, Med. 173
- , communicability of, to non-fever patients in hospital, discussed, Epid. 108
- , followed by cholesterol gall-stones containing typhoid bacilli (A. F. Hertz and F. S. Adams), Clin. 169
- , immunity to, paradox in, R.S.M. 196
- , inoculation against, R.S.M. 37, 38, 209
- , incidence on newcomers in districts, Epid. 180
- , in Belfast, causes of prevalence, Epid. 270-274
- , in Ipswich, diminution in prevalence, Epid. 111, 113, 116
- , in Leicester, diminution in prevalence, Epid. 111, 113
- , in London, diminution in prevalence, Epid. 112, 114, 115, 116, 118
- , in Manchester, causes of prevalence, Epid. 186, 187
- , death-rates in Manchester 1891-1906, Epid. 178, 179
- , influence of consumption of shell-fish on, Epid. 190, 191-209
- , onset of cases compared with deaths from diarrhœa (1891-1906), Epid. 192-206
- , in relation to prevalence of flies, Epid. 175, 178, 179

- Typhoid fever, transmission by house-flies, Epid. 189
 ——— in Nottingham, prevalent, Epid. 111, 115
 ——— in Wigan, prevalence stationary, Epid. 112, 114, 117
 ——— in Workington, Epid. 261
 ———, notification of, cases overlooked, Epid. 180
 ———, occurrence of cases in relation to prevalence of house-flies, Epid. 145-149
 ———, prevalence of, soil in relation to, Epid. 138
 ———, propagation by shell-fish, Epid. 182, 183, 184, 189, 190
 ———, rapid decay of teeth during, Odont. 77
 ——— in relation to infective arthritis, Baln. 93
 ———, seasonal prevalence and preceding rainfall, Epid. 137
 ———, severity of, dependent on oral sepsis, Odont. 101
 ———, sex incidence, preponderance of males over females, Epid. 164
 ———, slighter and overlooked cases most potent source of infection, Epid. 158
 ———, transmission by cockroaches, rats, and mice discussed, Epid. 209-211
 ——— by direct contact, Epid. 182
 ——— by house-flies, Epid. 138, 212-215, 262, 263, 265, 268
 ——— in India, Epid. 262, 263
 ——— in South Africa, Epid. 263
 ——— from one person to another, Epid. 182
 Typhoid vaccine, ingestion of, R.S.M. 81, 82, 93, 94
 ———, perpetual efficiency of cultures and subcultures, R.S.M. 164
 ———, therapeutic effects, R.S.M. 82-84, 209

 Udder, tuberculosis of, in relation to tuberculosis of other organs, Epid. 219, 220
 Uganda, natives of, effects of syphilization on, Neur. 45
 Ulcer of doubtful nature on the left arytaenoid cartilage (H. Betham Robinson), Laryng. 29
 ——— (tuberculous, large), on right side of tongue (H. J. Davis), Laryng. 81
 ———, see also *Rodent ulcer*
 Ulceration, vaccine treatment instead of formation of scar tissue, R.S.M. 14
 Ulcers (tuberculous), treatment with vaccine, results, R.S.M. 147
 Ultra-microscope, demonstration by, of living *Treponema pallidum* (*Spirochaeta pallida*) and various spirochaetes (H. W. Bayly), Clin. 3
 ———, detection of *Spirochaeta pallida* by, Surg. 238
 ———, value of, Clin. 4
 ———, diagnosis of syphilis by, Clin. 4
 ———, examination under, artificial light for, Clin. 5
 Ulna, subperiosteal resection of shaft for tuberculous osteomyelitis (C. P. Childe), Child. 158
 UNDERWOOD, ARTHUR S.—Some Egyptian skulls, Odont. 153; discussion (p. 154): Mr. F. J. Bennett, Mr. Hopewell-Smith, Mr. W. Rushton, Mr. Gabell, Mr. Ashley Densham, Mr. Underwood (reply)
 Unemployment, insurance against, Epid. 37
 Unidactyly, case of (R. Hutchison), Child. 41
 ———, hereditary transmission of, Child. 42
 Union hospitals, estimation of poverty by number of deaths from sickness occurring in, Epid. 2, 3
 ———, post-graduate demonstrations in, advocated, Epid. 35
 ———, statistics of, need for compilation, Epid. 3
 United States, electro-static machines in use in, Electr. 83, 84, 85
 ———, prophylactic value of tetanus antitoxin, proved in, Med. 40
 University College Hospital, statistics of pericardial effusion from (F. J. Poynton and S. A. Owen), Med. 129
 Unna, pathology of acne, Path. 172
 Uræmia, specimen of urinary organs from boy who died apparently from (J. Walter Carr), Child. 90

- Uremia, threatening, in chronic nephritis, treatment by hot-air bath, Therap. 147
- Uranium nitrate, determination of sodium phosphates in urine by, Med. 15, 16
- Urates, dominant factors in precipitation of, Baln. 73
- , solubility of, Baln. 73, 80
- , see also *Tophi, uratic*
- Urea, amount excreted by kidney depends on amount of protein eaten, Therap. 141
- Ureter, right, impaction of large calculus in lower end of (A. Carless), Clin. 63
- , see also *Calculus (ureteral)*
- Uric Acid Monthly*, misrepresentations by, Therap. 84
- Urinary organs, specimen of, from boy who died apparently from uræmia, diphtheritic membrane being found in trachea after death (J. Walter Carr), Child. 90
- tract, coli-bacillus infections of, vaccine treatment, R.S.M. 104, 105, 188
- Urine, acetone in, in recurrent or cyclic vomiting and starvation, Child. 79, 82, 87
- , acidity of, increased by acid phosphate of soda, Med. 21
- , analysis of, to determine amount of sodium phosphates in, Med. 15, 16
- , calcium oxalate in, in excess, Med. 1, 2; see also *Oxaluria*
- , extravasation of, Surg. 171
- , incontinence of, in women, treatment by electricity, Electr. 7, 13, 14, 30
- , —, nocturnal, treatment by electricity, Electr. 7, 12, 15, 30
- of normal individuals, positive pancreatic reaction absent in, Med. 181
- , pancreatic (Cammidge) reaction in, in 1,475 cases (P. J. Cammidge), Med. 163-184
- , —, experimental investigation into origin and cause (P. J. Cammidge and H. C. G. Semon), Path. 79-92
- , presence of *Bacillus pyocyaneus* in, in case of supposed muscular rheumatism, R.S.M. 178
- , reduction of Fehling's solution by, in pityriasis rubra, Derm. 61
- , residual, voidance of, by old men, important, Electr. 146
- , retention of, following intraspinal injection of magnesium-sulphate solution in treatment of tetanus, Med. 46
- , sodium chloride method of estimating, Baln. 82
- Urticaria, cases of (G. W. Dawson), Derm. 47
- pigmentosa, case of (G. N. Meachen), Derm. 108
- , case of very exceptional severity (E. G. Graham Little), Derm. 3
- Uterus, adenofibromatous polypus of, showing early malignant disease (J. H. Targett), Obst. 155
- , cancer of, course sometimes painless throughout, Obst. 5
- , carcinoma of body of, with keratinization of the growth (J. H. Targett), Obst. 153
- , cervix of, adenoma, malignant cystic (J. B. Hellier), Obst. 100
- , coli-bacillus infections of, vaccine treatment, R.S.M. 104
- , cystic tumour of right broad ligament springing from, and apparently developing from, Gartner's duct (A. H. N. Lewers), Obst. 67
- , diseases of, relation to rheumatoid arthritis, Baln. 105
- , treatment by electricity, Obst. 22
- , displacement backward, with adhesions, treatment by ventrofixation, Obst. 229, 230, 231, 233
- (double) (atresia of one-half), with dysmenorrhœa (A. H. N. Lewers), Obst. 241
- , with dysmenorrhœa, difficulty of diagnosis, Obst. 242
- , fibroid of cervix and body of uterus (J. P. Hedley), Obst. 189
- , fibroids of treatment by radium, Electr. 71
- , fibromyoma of, life-history of (A. J. Wallace), Obst. 208-220
- , life-history, table of cases illustrating, Obst. 216, 217
- , fibrosis of, causing persistent menorrhagia (J. Inglis Parsons), Obst. 240; report of Pathology Committee, p. 241
- , malignant tumour, atypical (H. J. Paterson), Obst. 99; report on specimen by Pathology Committee, p. 99

- Uterus, myomatous tumour of, simulating sarcomatous growth (H. Macnaughton-Jones), Obst. 103; report on specimen (C. Lockyer), p. 104
 — (prolapsed), carcinoma of cervix of, in patient aged 86, with contact carcinoma on one of the labia; vaginal hysterectomy (H. R. Andrews), Obst. 161
 — —, case with severe cardiac disease operated on under local and spinal anaesthesia (J. Inglis Parsons and T. C. Clare), Obst. 104
 — —, uncomplicated, with retroflexion, treatment, Obst. 228
 — of rabbit, result of double oöphorectomy upon growth of (S. G. Shattock), Path. 51
 —, rupture of, treated by suture of rent *per vaginam* (H. J. Paterson), Obst. 58
 — —, spontaneous transverse, at fundus during labour (A. H. N. Lewers), Obst. 136
 —, sarcoma of, when painful, Obst. 8
 — —, of the body of, from a girl aged 20 (H. Jellett), Obst. 186
 — (septate), pregnancy in (W. Tate), Obst. 190
 — — —, treated by abdominal hysterectomy (W. Tate), Obst. 190
 —, subinvolution, Obst. 225
 — — —, treatment by electricity, Electr. 7, 8, 11, 22
 — — —, palliative, Obst. 226
 — — —, of, with displacement of ovaries, Obst. 226, 227
 — — — — —, treatment, Obst. 227, 228
 —, suspension of, compared with ventrofixation, Obst. 234
 —, torsion of body, specimen (W. S. A. Griffith), Obst. 18
 —, tumour of, of doubtful nature (J. Barris), Obst. 73
 — — —, treatment by electrolysis, method of Apostoli scientific, Electr. 4, 5
 Uvula, malformation of, case (J. D. Grant), Laryng. 131
 —, papillomata of (F. Coleman), Odont. 20

Vaccination, hypothesis of, in X-ray and radium irradiation, R.S.M. 156

Vaccine therapy, aim of, to stimulate patient to produce own protective substances, R.S.M. 93

- — — in abscess of liver, R.S.M. 57
 — — — in acne, R.S.M. 95, 184, 188
 — — — in acute bronchitis, R.S.M. 63
 — — — — coli infection of bladder, R.S.M. 73
 — — — — cystitis, R.S.M. 184, 185
 — — — in adenoids, R.S.M. 215
 — — — in appendicitis, unsuccessful case, R.S.M. 185
 — — — in *Bacillus coli* infections, R.S.M. 104, 105, 188
 — — — in bacterial diseases of mouth, R.S.M. 87, 88
 — — —, bias in favour of or against, R.S.M. 34, 35
 — — — in boils, R.S.M. 65, 95, 103, 188
 — — — in bronchiectasis, R.S.M. 59, 114, 115
 — — — in cancer, R.S.M. 18
 — — — of cancer, scientifically not justifiable, Therap. 78, 79
 — — —, capable of regulation, R.S.M. 162
 — — — in case of invasion by *Bacillus coli*, R.S.M. 57
 — — —, cautious mode of advance in, R.S.M. 79
 — — — in chronic bronchitis, R.S.M. 146
 — — — tuberculosis, limitations, R.S.M. 148
 — — — — —, results, R.S.M. 147
 — — —, clinical observation as guide to, R.S.M. 124
 — — — in colitis, R.S.M. 104, 105
 — — — in conjunctivitis, R.S.M. 62
 — — — in dentistry, R.S.M. 86; see also *Pyorrhæa alveolaris*
 — — — in diabetes, R.S.M. 18
 — — —, difficulties in, R.S.M. 144

- Vaccine therapy, discussion on, R.S.M. 1-216: opened by Sir A. E. Wright, continued by Dr. Charles Slater, Dr. L. Colebrook, Dr. StClair Thomson, Dr. Hale White, Dr. J. W. Eyre, Dr. Harold A. des Vœux, Mr. H. W. Bayly, Dr. William Bulloch, Sir William B. Leishman, Dr. R. T. Hewlett, Mr. K. W. Goadby, Dr. J. Henderson Smith, Dr. A. Whitfield, Dr. John Freeman, Dr. A. Butler Harris, Dr. J. Kingston Fowler, Dr. Arthur Latham, Mr. S. Maynard Smith, Dr. Alexander Fleming, Dr. T. J. Horder, Dr. D. W. Carmalt-Jones, Dr. F. Rufenacht Walters, Mr. W. Deane Butcher, Dr. H. Lewis Jones, Dr. G. A. Crace-Calvert, Mr. Leonard Noon, Dr. W. Parry Morgan, Mr. J. Courtenay MacWatters, Mr. F. Ashton Warner, Dr. W. d'Este Emery, Dr. David Lawson, Dr. A. C. Inman, Dr. P. Watson Williams
- in eczema, R.S.M. 103, 183
 - in empyema, R.S.M. 58
 - of endocarditis, Therap. 104, 105, 114, 115
 - , failure of, cases apparently pointing to, R.S.M. 35, 36
 - , illustrating, R.S.M. 49
 - in general practice, R.S.M. 102
 - in septicæmia, R.S.M. 59, 60
 - in genito-urinary diseases, R.S.M. 63
 - in glanders, R.S.M. 66
 - in glycosuria, R.S.M. 178-181
 - in gonococcal arthritis, R.S.M. 55-57, 183, 188, 189
 - in gonococcal iritis, R.S.M. 189
 - in gonorrhœal ophthalmia, R.S.M. 62
 - , caution in application necessary, R.S.M. 62
 - , guides in, R.S.M. 211
 - in infective endocarditis, reason for failure, R.S.M. 119
 - in inflammation at roots of teeth, and toothache, R.S.M. 15
 - in influenza, R.S.M. 69-71, 73, 185
 - knowledge of, required by medical practitioner, R.S.M. 2
 - , limitations of, R.S.M. 11-14, 102, 108, 113, 118-128, 143, 212
 - in lupus, results, R.S.M. 147
 - , mechanism, alterative action, R.S.M. 195
 - , local reaction, R.S.M. 192
 - , production of immunity, R.S.M. 196
 - , methods complicated and difficult, R.S.M. 78
 - in middle-ear disease, R.S.M. 160
 - in orbital cellulitis, R.S.M. 62
 - in pneumonia, R.S.M. 103, 104
 - , prophylactic, R.S.M. 85
 - in prophylaxis of bacterial infection in cases of individual susceptibility, R.S.M. 19
 - in pruritus ani, R.S.M. 15
 - in puerperal septicæmia, R.S.M. 59, 63
 - in pyæmia, R.S.M. 100, 101
 - in pyorrhœa alveolaris, R.S.M. 62, 172, 148, 149; Odont. 36, 38
 - (early cases), Odont. 55
 - (J. W. Eyre and J. L. Payne), Odont. 29
 - , radio-active and electric methods, proposal that Sections of the Society interested should combine to study, R.S.M. 156
 - , rationale of, R.S.M. 1
 - , recurrences after, R.S.M. 144
 - in relation to diagnosis of pulmonary tuberculosis, R.S.M. 204
 - , repeated inoculation in, R.S.M. 22
 - , results in local and general infections compared, R.S.M. 66
 - sometimes uncertain, R.S.M. 147
 - in rheumatism due to chronic suppurative lesions in mouth, R.S.M. 89, 90

- Vaccine therapy in sinusitis, R.S.M. 215
 ——— in staphylococcic infection, R.S.M. 67
 ——— still in experimental stage, R.S.M. 211
 ——— in suppuration of nasal accessory sinuses, R.S.M. 52-53
 ——— of syphilis, Surg. 229
 ——— in tuberculosis of conjunctiva, R.S.M. 61
 ——— in tuberculous arthritis, R.S.M. 129-136
 ——— in ulceration, instead of formation of scar tissue, R.S.M. 14
 ——— in ulcerative colitis, R.S.M. 58
 ———, value in diagnosis, R.S.M. 63, 64
 ——— of, R.S.M. 33-37, 75
 ———, admission or rejection of evidence for, R.S.M. 39, 40, 41
 ———, determined by animal experiments with control series, R.S.M. 140
 ——— by bacteriological tests, R.S.M. 39-41
 ——— by scientific methods desirable, R.S.M. 140
 ——— of opsonic index in, R.S.M. 44, 45, 95, 96
 ———, unsettled opinions as to, R.S.M. 109
 ——— in whooping-cough, R.S.M. 97-100
 ——— in X-ray dermatitis, R.S.M. 14
- Vaccines, administration of, control by opsonic index, R.S.M. 32, 117, 142
 ———, mode of, R.S.M. 37, 141
 ———, hypodermic, R.S.M. 37, 73, 74, 79, 85, 118, 211
 ———, oral, R.S.M. 37, 73, 74, 79, 84, 92, 93, 94, 117, 118, 182, 198, 211
 ———, in pill form, R.S.M. 93
 ———, proof of absorption, R.S.M. 117
 ———, rectal, R.S.M. 37
 ———, to whom to be administered, R.S.M. 143, 210
 ———, dosage, R.S.M. 32, 47, 93, 96, 98, 116, 142, 165, 166
 ———, opsonic index failing as guide to, R.S.M. 98, 121
 ———, effects of, tested by inoculation into rabbits, R.S.M. 81
 ———, proof of causal relationship of organisms before used as, R.S.M. 97
 ———, properties that should be possessed by, R.S.M. 161, 162
 ———, virulence of, standard for, lacking, R.S.M. 144
 ——— (autogenous) more effective than stock vaccines, R.S.M. 119
- Vacuum electrode, application in static electricity in treatment of acne vulgaris, Electr. 85, 86
 ——— of facial flushing, Electr. 82
- Vagina, adenomyoma of wall of (T. G. Stevens), Obst. 57
 ———, diseases of, treatment by electricity, Obst. 19
 ———, fibroid of wall of (H. Russell Andrews), Obst. 129
 ———, method of introducing radium into, in treatment of cancer, Electr. 62
 ———, suture of rupture of uterus through (H. J. Paterson), Obst. 58
- Van den Akker, first performance of Cæsarean section in eclampsia by, Obst. 196
- Vascular and verrucose elements (combined) accompanying linear nævus (H. G. Adamson), Derm. 103
- VEALE, R. A., and HANN, R. G.—Fatal case of poisoning by phosphorus, taken as an abortifacient, with unusual subcutaneous hæmorrhages, Therap. 64
- Vegetables, calcium oxalate contained in, Med. 4
- Veins in neck, distension of, as signs of pericardial effusion, Med. 87, 88
- Velpeau, rupture of tunica vaginalis in hydrocele, quoted, Surg. 166, 171
- Veneral diseases in British Army, Advisory Board reports on, Surg. 225
- Venous obstruction in case of cyanosis (W. P. Herringham), Clin. 168
- Ventnor Sanatorium, records of tuberculous infection of staff, Med. 139
- Ventrofixation in treatment of backward displacement of uterus, with adhesions, Obst. 229, 230, 231, 233, 234
 ——— compared with suspension, Obst. 234

- Venus fly-trap, reflex action of, Path. 29
- Veratrum in treatment of puerperal eclampsia, Obst. 205, 206
- Verrucose and vascular elements (combined) accompanying linear nævus (H. G. Adamson), Derm. 103
- Version in contracted pelvis, Obst. 115, 120
- Vertebræ, lumbar, tuberculous infection of, treatment by tuberculin, with aid of opsonic index, R.S.M. 48
- , normal individual percussion, notes of, Med. 223
- , —, —, —, —, diagnostic application, Med. 223
- Vertebrata, cancer limited to, Laryng. 159
- , cancer ubiquitous among, Therap. 70
- , continuity of neurones in, discussed, Path. 27, 28
- Vertigo, accompanying cases of tumour of pituitary body, Neur. 73, 74
- (laryngeal), case of (H. J. Davis), Laryng. 79
- , epileptiform attacks accompanying, Laryng. 81
- , treatment by administration of drugs, Laryng. 80
- Vestibular reactions, active, accompanying hysterical deafness (P. H. Abercrombie and D. McKenzie), Otol. 74
- DES VŒUX, HAROLD.—Discussion on vaccine therapy, R.S.M. 64
- Victoria Hospital for Children, Chelsea, statistics of pericardial effusion from (H. F. L. Hugo), Med. 119
- Victoria Park Hospital, records of tuberculous infection of staff, Med. 139
- Villi, oedema of connective tissue of, in case of tubal mole (R. D. Maxwell), Obst. 235
- VINCENT, RALPH.—Discussion on communication of diarrhoea from sick to healthy, Epid. 122
- , discussion on lymphatism, Anæsth. 49
- Vinegar, consumption of, factor in production of gastric ulceration, Therap. 132
- VINRACE, DENNIS.—Discussion on lupus of nose and throat, Laryng. 87
- Vinsonneau, experiments on power of resistance of tunica vaginalis, quoted, Surg. 167
- Violet-flame discharge in static electricity, effect on screen, Electr. 78
- , —, —, —, —, therapeutic effects, Electr. 76
- Virchow, R., catarrhal jaundice, quoted, Med. 171
- , histology of osteo-arthritis, quoted, Baln. 90
- Visual discharges from organic disease, Neur. 11
- Vital statistics of Manchester (1901-1903), Epid. 17
- of Naples, Baln. 25
- Vittel, waters of, diuretic action explained, Baln. 9
- Vocal cord (left), epithelioma of, removal by thyrotomy (J. Dundas Grant and D. McKenzie), Laryng. 19
- , —, —, specimen of larynx showing fresh outbreak on, after removal of right cord for epithelioma, September, 1896 (H. Tilley), Laryng. 33
- , —, —, infiltration of ventricular band, tuberculosis? neoplasm? (J. Dundas Grant), Laryng. 16
- , —, —, and inter-arytenoid space, tuberculosis of, completely healed by two months' silence and sanatorium treatment (StC. Thomson), Laryng. 6
- , —, —, partial fixation of twenty-one years' duration (I. Moore), Laryng. 19
- , —, —, (right), epithelioma of case shown seven months after operation, no recurrence, development of cicatricial substitute for vocal cord (J. Dundas Grant), Laryng. 156
- , —, —, —, removal by thyrotomy (J. Dundas Grant), Laryng. 17
- , —, —, —, paralysis of, in myotonia atrophica (H. Clayton Fox), Laryng. 20
- Vocal cords, tuberculosis of, cicatrized with seven applications of galvano-cautery (StC. Thomson), Laryng. 6
- Vohsen's method of lateral pharyngotomy in removal of lymphosarcoma of tonsil (G. Wilkinson), Laryng. 86
- Voice, loss of, with dyspnoea (W. Stuart-Low), Laryng. 162
- (singing), impairment of, in hypertrophy of lingual tonsil, improved by snaring operation (J. Dundas Grant), Laryng. 155

- Voice-training, Laryng. 32, 50
Voice-users, difficulties of, Laryng. 42, 52
v. Volkmann, differentiation of rheumatoid and osteo-arthritis by, Baln. 86
Volvulus, death from, in case of chylous cyst of mesentery associated with enlarged glands in mesentery (H. Burrows), Child. 164
Vomit in gastric cancer, chemical changes in, Surg. 136
Vomiting accompanying tumours of pineal body, Neur. 72
— in early phthisis, R.S.M. 17
—, entirely absent in case of temporo-sphenoidal abscess (A. L. Whitehead), Otol. 73
— in gastric ulcer, Med. 119
— — —, Surg. 134
— — — (chronic), treatment, Therap. 130
— in intussusception, Child. 167
— (persistent), due to disease of kidney (A. D. Fordyce), Child. 1, 6, 10
— (post-operative), in cases of duodenal ulcer, Surg. 86
— of pregnancy, treatment by static wave current, Electr. 107
— (recurrent or cyclic), accompanied by acute starvation, Child. 82
— — —, association with hypertrophic stenosis of pylorus, case of (A. E. Russell), Child. 78
— — — — —, autopsy, Child. 80
— — — —, fatty degeneration of liver in, Child. 83
— — — —, muscular spasm in hypertrophic stenosis of pylorus as factor in, Child. 85
— — — —, pathology of, current views on, Child. 81
— — — —, presence of acetone in breath and urine, Child. 79, 82, 87
— — — —, treatment, indications for, Child. 85
—, stomach-ache and screaming in infants, common symptoms masking early onset of intussusception, Child. 167
VOORHUIS, J. A.—On the therapeutical use of static electricity, Electr. 73-81; discussion (p. 82): Dr. Sloan, Dr. Curtis Webb, Dr. Lewis Jones, Dr. F. H. Humphris, Dr. Pirie, Dr. Reginald Morton (reply)
Vulsellum-catch forceps for firmly securing submerged tonsils in the operation for their more efficient removal (H. Tilley), Laryng. 124
Vulva, carcinomata of (multiple), Obst. 45
—, diseases of, treatment by electricity, Obst. 19
—, kraurosis of, definition, Obst. 35
— — —, pathology of, Obst. 36
— — —, with commencing carcinoma (A. E. Giles), Obst. 54
— — — and carcinoma of, leucoplakic vulvitis in relation to (C. Berkeley and V. Bonney), Obst. 29
—, see also *Pruritus, vulvar and anal*
Vulvitis (leukoplakic), (C. Berkeley and V. Bonney), Obst. 29
— — —, etiology, Obst. 39
— — —, age incidence, Obst. 38
— — —, definition, Obst. 30
— — —, pathology, Obst. 31
— — —, relation to kraurosis vulvæ and carcinoma vulvæ (C. Berkeley and V. Bonney), Obst. 29

WAGGETT, E. B.—Discussion on case of audible tinnitus, Otol. 2
—, discussion on chronic middle-ear suppuration, Otol. 44
— — — on papillomata of larynx from a boy aged 6, removed by direct method, Laryng. 98
WAGSTAFF, E.—Discussion on the static wave current, Electr. 106
Walcher's position to assist fixation of head in brim of pelvis, Obst. 114, 120, 127
Waldeyer, terms of neurone concept, quoted, Path. 1-2

- WALKER, J. W. THOMSON.—Case of resection of bladder for malignant disease, *Clin.* 68
 —, discussion on case of impaction of large calculus in lower end of right ureter, *Clin.* 64
- WALL, CECIL.—Tabes with amyotrophy, *Neur.* 27; discussion (p. 28): Dr. Wall, Dr. Harris, Dr. Wall (reply)
- WALLACE, A. J.—Contribution to the life-history of fibromyomata of the uterus, *Obst.* 208-220; discussion (p. 220): Dr. Herbert Spencer, Dr. Amand Routh, Dr. Macnaughton-Jones
- Wallace, C. S., double oöphorectomy, quoted, *Path.* 51
- WALLACE, J. SIM.—Discussion on susceptibility of teeth to dental caries, *Odont.* 81
 —, susceptibility of teeth to caries, quoted, *Odont.* 78, 79
- Waller, origin of nerve fibres, *Path.* 2
- WALLIS, C. E.—Discussion on condition of the mouth in chronic disease, *Odont.* 105
 — — on radium in treatment of malignant growths, *Electr.* 69
- Walsham, H., tuberculous infection of tonsils, quoted, *Child.* 28
- WALTERS, F. RUFENACHT.—Discussion on vaccine therapy, *R.S.M.* 149
- WARNER, F. A.—Discussion on vaccine therapy, *R.S.M.* 182
- WARRINGTON, W. B.—Discussion on distal type of myopathy, *Neur.* 94
- Warthin, on question of thymus death, quoted, *Child.* 47
- Warts, refrigeration in treatment of, *Electr.* 44
- Washbourn, J. W., bacteriology of pyorrhæa alveolaris, quoted, *Odont.* 39
- Wassermann, Professor, complement-fixing test in diagnosis of syphilis, *Surg.* 239
- Wassermann's reaction, *Neur.* 62, 63
 — —, destruction by mercury treatment, *Surg.* 221, 229, 230, 235
 — —, examination of blood-serum of idiots by (H. R. Dean), *Neur.* 117
 — — of pregnant women by, *Neur.* 121
 — — of cerebrospinal fluid of idiots by, *Neur.* 118, 123
 — —, importance of, illustrated by case of syphilis (J. E. R. McDonagh), *Derm.* 90
 — — in diagnosis of general paralysis, positive, *Neur.* 40-42, 61
 — — of syphilis, *Derm.* 40
 — —, *Surg.* 220, 227, 229, 231, 232, 243
 — —, inconclusive or negative in primary stages, *Surg.* 222, 223
 — — (modification), in diagnosis of syphilis, *Surg.* 239, 240
 — — in diagnosis of active syphilitic disease of nervous system, negative, *Neur.* 61
 — — of tabes, *Neur.* 40-42
 — —, positive, diagnostic of syphilis, even in absence of signs and symptoms, *Neur.* 120
- Wasting infants fed on undiluted citrated milk (F. Langmead), *Child.* 104
 — in gastric ulcer, *Therap.* 119
- Water-carriage system of sewerage, effect on diarrhœa, *Epid.* 111, 118
- Water-closet system established in London, *Epid.* 111, 112
 — — prevalent in Ipswich, *Epid.* 111, 115
 — — in Leicester, *Epid.* 111
- Water-supply of Naples, improvements in, *Baln.* 22, 23
- WATERHOUSE, H. F.—Discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 91
- Waters, radio-active, ingestion produces enlargement of thyroid gland, *Baln.* 9
- WATSON, C. GORDON.—Case of tibio-calcaneal resection at the ankle, with amputation at the foot, *Clin.* 133; discussion (p. 134): Mr. A. E. Barker, Mr. C. G. Watson (reply)
 —, discussion on arthritis deformans, *Baln.* 96
 — — on case of (?) hyperplastic tuberculosis of pelvic colon, *Clin.* 206
- Watson, Chalmers, on pancreatic reaction, quoted, *Med.* 204
- Wax-plate method of Born, used in reconstruction model showing auditory apparatus of 16 mm. human embryo (G. J. Jenkins), *Otol.* 34
- Weakness, profound, clinical indication of diagnosis of descending thoracic aorta, *Clin.* 126
- WEBB, J. CURTIS.—Discussion on therapeutical use of static electricity, *Electr.* 83
 —, discussion on the static wave current, *Electr.* 106

- WEBB, J. CURTIS.—Points for the use of, and indications for, electrotherapy in some gynæcological affections, *Obst.* 16; discussion (p. 25); Dr. Lewis Jones, Mr. Spanton, Dr. Amand Routh, Dr. Herman Stedman, Dr. F. Howard Humphris, Dr. Macnaughton-Jones
- WEBER, F. PARKES.—Ateleiosis in a man aged 42; physical development said to have been arrested at about the age of 9, *Child.* 143; discussion (p. 146): Dr. Shuttleworth, Dr. J. W. Carr, Dr. F. Parkes Weber (reply)
- , bile-producing primary malignant tumour of liver (malignant adenoma), with remarks on the cells of malignant tumours retaining some of the metabolic or other functional characteristics of the cell-type from which they are derived, *Path.* 147
- , case of Recklinghausen's disease, *Clin.* 79
- , chronic arteritis obliterans (lower extremity) associated with phlebitis, *Clin.* 97
- , congenital tumour of the left parotid region (? cavernous lymphangioma), *Clin.* 144; discussion (p. 144): Mr. A. Pearce Gould
- , discussion on air-swallowing, *Child.* 152
- , on cases of adiposis dolorosa, *Clin.* 61
- , of angioneurosis, *Clin.* 106
- , on case of bending of bones of both legs and partial ankylosis of both hips, *Clin.* 95
- , of congenital acholuric jaundice, *Child.* 137
- , enlargement of one limb in brother and sister, *Child.* 44
- , hemidystrophy, *Child.* 23
- , œdema of leg, *Child.* 89
- , on two cases of congenital œdema with cardiac defect in mother and daughter, *Clin.* 67
- , on case of cyanosis, *Clin.* 169
- , for diagnosis, *Derm.* 88
- , of hereditary multiple telangiectases and on case of lymphangioma of tongue, *Clin.* 111
- , of jaundice with areas of unpigmented skin, *Clin.* 197
- , of myelogenous leukæmia in infant, *Child.* 94
- , on cases of oxycephaly, *Child.* 128
- , on case of pigmentation of circum-oral skin and of buccal mucosa, *Clin.* 9
- , of rheumatoid arthritis with albuminuria, and with great enlargement of liver, *Clin.* 62
- , on cases of subacute liver atrophy, persistent vomiting, and lymphosarcoma, *Child.* 13
- , on case of xeroderma pigmentosa with unusual features, *Derm.* 58
- , on cervical ribs, *Clin.* 156
- , on congenital pulmonary stenosis, *Child.* 151
- , on extensive myositis ossificans, *Clin.* 154
- , on primary hyperplastic tuberculosis of stomach and duodenum, *Clin.* 43
- , on pyæmic knee- and shoulder-joints treated by tapping and incision, *Clin.* 121
- , on specimen of heart of infant showing nodules on endocardium, *Child.* 46
- , on spleno-medullary leukæmia, *Clin.* 138
- , on thoracic aneurysm in a boy, *Clin.* 36
- , exophthalmic goitre in a man with symmetrical telangiectases of the ocular conjunctivæ, *Clin.* 142
- , family amaurotic idiocy without characteristic ophthalmoscopic signs, *Child.* 58
- , intermittent claudication due to chronic arteritis obliterans, in a tobaccoist, *Clin.* 96
- , internal hydrocephalus and amaurosis without definite ophthalmoscopic changes, following symptoms of posterior basic meningitis or ependymitis, *Child.* 62
- , patient a few months after omentopexy and peritoneal drainage for chronic ascites connected with hepatic cirrhosis, *Clin.* 80
- , splenomegaly and hydrocephalus, *Child.* 63
- , and MICHELIS, E.—Disappearance of chronic ascites in alcoholic cirrhosis after repeated paracentesis and laparotomy without omentopexy, *Clin.* 167

- WEBER, F. PARKES, and NORMAN, R. H.—Bluish pigmentation of the skin, *Clin.* 140; discussion (pp. 141-142): Dr. C. F. Martin, Dr. J. Galloway, Dr. T. Thompson, Dr. H. D. Rolleston, Dr. F. Parkes Weber (reply)
- , case of argyria, *Derm.* 75
- Webster, J. Clarence, cause of tubal pregnancy, quoted, *Obst.* 238
- “Weeping blood” condition, *Surg.* 195, 202, 205
- Weil, P., and Boidin, acute meningitis during secondary syphilis, quoted, *Med.* 37
- WELLS, PRIMROSE.—Discussion on electrical treatment of prostatic eruption, *Electr.* 147
- Wendover, medical history of, *Baln.* 42
- WEST, C. ERNEST.—Case of ? congenital defect of both tympanic plates, *Otol.* 61
- , discussion on deformity of both pinnae, *Otol.* 72
- , pathology of cholesteatomata, *Otol.* 69
- , on pedunculated papilliform growth springing from the posterior border of the cartilaginous meatus, *Otol.* 46, 47
- , on post-operative tests in labyrinthine disease, *Otol.* 7
- , on right external meatus, *Otol.* 76
- WEST, SAMUEL.—Pericardial effusion, its diagnosis and treatment: an address introductory to a discussion on the subject, *Med.* 55-73 (reply) *Med.* 102
- WESTERN, G. T.—Series of cases of scrofuloderma and lupus treated by tuberculin injections, *Derm.* 8
- WESTMACOTT, F. H.—Discussion on early symptoms in case of epithelioma originating near right Eustachian tube, *Otol.* 31
- , discussion on exostoses of right external meatus, *Otol.* 78
- , on obstruction of both nostrils, *Laryng.* 149
- , on party-wall pharyngeal cancer, *Laryng.* 143
- Westminster Hospital, statistics of pericardial effusion from (A. M. Gossage and H. F. Marris), *Med.* 110
- Westphal, organic disease, cause of olfactory discharges, quoted, *Neur.* 2, 4
- WETHERED, F. J.—Discussion on hospital infection of tuberculosis, *Med.* 161
- WHEATON, S. W.—Discussion on summer diarrhoea and enteric fever, *Epid.* 269
- WHIPHAM, T. R.—Case of air-swallowing, *Child.* 151; discussion (p. 152): Dr. Parkes Weber
- , case of congenital malformation of the heart, *Child.* 149
- , of congenital stenosis in a girl aged 18, *Child.* 150; discussion (p. 150): Dr. Carr, Dr. Hawkins, Dr. Parkes Weber, Dr. Cautley, Dr. Whipham (reply)
- , discussion on case of chloroma, *Child.* 20
- , myelogenous leukemia in an infant aged 18 months, *Child.* 92; discussion (p. 94): Dr. Cautley, Dr. Parkes Weber, Dr. D. Forsyth, Dr. Whipham (reply)
- , pathological report on case of myelogenous leukemia in an infant aged 18 months, *Child.* 152
- WHITE, CLIFFORD.—Quadruplets born at the twenty-sixth week, *Obst.* 79
- White, C. Powell, formation of epithelial metaplasia, *Otol.* 66
- White, Gilbert (of Selborne), on characteristics of eunuchs, quoted, *Path.* 120
- White, Sinclair, proportion of ureteral to renal calculus, quoted, *Electr.* 94
- WHITE, W. HALE.—Discussion on arthritis deformans, *Baln.* 96
- , discussion on diagnosis and treatment of duodenal ulcer, *Surg.* 89
- , on thoracic aneurysm in a boy, *Clin.* 37
- , on vaccine therapy, *R.S.M.* 55
- , gastrostaxis, quoted, *Surg.* 195, 205
- , on mucous colitis, alluded to, *Therap.* 60
- , BRUCE-PORTER, H. E., and MUMMERY, J. P. LOCKHART.—Case of pneumococcic colitis with hyperpyrexia: severe hemorrhage treated by appendicostomy, *Clin.* 48; discussion (p. 51): Mr. Lockhart Mummery, Dr. Dalton, Mr. Symonds, Dr. Hale White (reply)
- WHITEHEAD, A. L.—Case of temporo-sphenoidal abscess in which vomiting was entirely absent, *Otol.* 73; discussion (p. 74): Dr. Milligan, Mr. Whitehead (reply)
- , discussion on anatomical and pathological preparations, *Otol.* 26

- WHITEHEAD, A. L.—Discussion on case of audible tinnitus, Otol. 2
 —, discussion on chronic middle-ear suppuration, Otol. 43
 — — on early symptoms in case of epithelioma originating near right Eustachian tube, Otol. 31
 — — on exostosis of right external meatus, Otol. 77
 — — on post-operative tests in labyrinthine disease, Otol. 7
 — — on probable late secondary specific pharyngitis, Laryng. 155
- WHITFIELD, A.—Case of (?) mycosis fungoides, Derm. 63: discussion (p. 64): Dr. Pringle, Dr. Colcott Fox
 —, discussion on case of *kératodermie blennorrhagique*, Derm. 80
 — — of lichen planus annularis, Derm. 67
 — — of lichen planus in a diabetic, Derm. 46, 47
 — — of onychia sicca syphilitica, Derm. 44
 — — of syphilitic erythema nodosum, Derm. 51
 — — on curious case of affection of finger- and toe-nails, Derm. 7
 — — on importance of the Wassermann reaction, Derm. 93
 — — on two cases for diagnosis, Derm. 70, 72
 — — on vaccine therapy, R.S.M. 94
 —, peculiar form of pigmentation of the face and neck in a woman aged 40, Derm. 76
 —, some points in the pathology of acne, Path. 172
- Whitlow, case of, tuberculous nature diagnosed, R.S.M. 95
- Whooping-cough, bacillus of, R.S.M. 97, 98
 —, treatment by saline-solution injections, R.S.M. 99, 100
 — — by vaccine, R.S.M. 97-100, 121
- Widal and Javal, on excretion of salt from and retention in body, Baln. 67, 68
- Wigan, diarrhoea in, prevalence stationary, Epid. 112, 114, 117, 118, 119
 —, pail-closet system prevalent in, Epid. 111, 112, 113
 —, prevalence of summer diarrhoea in, Epid. 259
 —, typhoid fever in, prevalence stationary, Epid. 112, 114, 117
- Wiggin, statistics of intussusception in children, Child. 170, 172
- Wildungen, waters of, diuretic action explained, Baln. 9
- WILKINSON, GEORGE.—Discussion on case of tabetic deafness, Otol. 39
 — — on hypertrophic laryngitis with stenosis in syphilitic subject, Laryng. 93
 —, rare congenital deformity of the nose in an infant, Laryng. 83: discussion (p. 85): Dr. Watson Williams, Dr. W. Hill, Dr. Dan McKenzie, Dr. Pegler, Dr. Dundas Grant, Mr. Wilkinson (reply)
 —, pulsating angioma (angioma racemosa, cirroid aneurysm) of the right auricle, Otol. 35
 —, specimen of lymphosarcoma of the tonsil, removed by lateral pharyngotomy after Vohsen's method, Laryng. 86
 —, two cases of cholesteatoma of unusual size extending into the posterior fossa and causing obliteration of the sigmoid sinus, Otol. 82
- WILKINSON, W. CAMAC.—Discussion on primary hyperplastic tuberculosis of stomach and duodenum, Clin. 43
- Willcox, W. H., and Morgan, W. P., opsonic index of pneumococcus, quoted, R.S.M. 121
- WILLETT, J. ABERNETHY.—Placenta and membranes of quadruplets, Obst. 80: discussion (p. 80): Dr. R. H. Hodgson
- WILLEY, FLORENCE.—Discussion on very large cervix fibroid, Obst. 185
- WILLIAMS, A. W.—Case of canities, Derm. 110
 —, case of Darier's disease, Derm. 95
 —, and GALLOWAY, JAMES.—Lichen planus in a diabetic, Derm. 45: discussion (p. 46): Dr. Galloway, Dr. Adamson, Dr. Whitfield, Dr. Williams (reply)
- WILLIAMS, C. THEODORE.—Discussion on hospital infection of tuberculosis, Med. 157
- WILLIAMS, GWYNNE.—Case of pulsating exophthalmos, Clin. 171
- WILLIAMS, H. LLOYD.—Discussion on unilateral overgrowth of lower jaw, Odont. 50
 — — on spindles of von Ebner, Odont. 151

- Williams, Leonard, action of saline spas, quoted, *Baln.* 78
 —, discussion on cases of adiposis dolorosa, *Clin.* 59
 — on lymphatism, *Anaesth.* 43
 — on treatment of stricture of rectum and other conditions by fibrolysin, *Clin.* 89
- WILLIAMS, P. WATSON.—Case of extrinsic malignant growth of larynx, *Laryng.* 145
 —, case of laryngeal new growth for diagnosis, *Laryng.* 60; discussion (p. 61): Dr. Dundas Grant, Dr. W. Hill, Dr. Scanes Spicer, Mr. Herbert Tilley, Dr. StClair Thomson, Dr. Watson Williams (reply)
 —, discussion on case of hoarseness of one month's duration in a woman aged 60, *Laryng.* 32
 — of hoarseness in woman, *Laryng.* 56
 — of laryngeal paralysis, *Laryng.* 151
 — of paresis of palate, pharynx, and oesophagus, *Laryng.* 91
 — of rare congenital deformity of nose, *Laryng.* 85
 — endothelioma of ethmoid, *Laryng.* 66
 — on infiltration of right half of larynx, *Laryng.* 147
 — on laryngeal vertigo, *Laryng.* 79, 81
 — on large tuberculous ulcer of right side of tongue, *Laryng.* 82
 — on papillomata of larynx from a boy aged 6, removed by direct method, *Laryng.* 97
 — on partial fixation of left vocal cord of twenty-one years' duration, *Laryng.* 20
 — on radical frontal sinus operation, *Laryng.* 144
 — on removal of a rapidly-growing soft fibroma from posterior wall of left maxillary antrum by a modified Rouge's operation, *Laryng.* 96
 — on sanatorium, silence, and galvano-cautery in treatment of tuberculosis of throat, *Laryng.* 9
 — on specimen of larynx showing the redevelopment of epithelioma on left cord after removal of right for same cause, September, 1896, *Laryng.* 35
 — on treatment of case of tuberculosis of larynx, *Laryng.* 153
 — on tumours of post-nasal space, *Laryng.* 161
 — on vaccine therapy, *R.S.M.* 215
 — on ulcer of doubtful nature on left arytenoid cartilage, *Laryng.* 29
 —, malignant growth of the right temporal bone, with extension through the external meatus, resembling an aural polypus, *Obst.* 85; pathological report by Professor W. Hall (p. 87)
- WILLIAMSON, HERBERT.—Discussion on Cæsarean section in placenta prævia, *Obst.* 14
 —, and BARRIS, J. D.—Abscess of the ovary probably due to infection by the *Amœba coli*, *Obst.* 152; discussion (p. 153): Dr. C. Lockyer
- WILLIAMSON, OLIVER K.—Spastic diplegia with mental defect, *Child.* 70
- WILLS, W. K.—Case for diagnosis, *Derm.* 12; discussion (p. 14): Dr. Colcott Fox, Dr. J. M. H. MacLeod, Dr. Little, Dr. Wills (reply)
- WILSON, C.—Discussion on examination of intestines by X-rays, *Electr.* 122
- WILSON, S. A. K.—Discussion on case of olivo-ponto-cerebellar atrophy, *Neur.* 91
 —, and WOOD, G.—Two cases of a nervous disease of undetermined nature occurring in a brother and sister, *Neur.* 96; discussion (p. 98): Dr. H. Head, Dr. S. A. K. Wilson (reply)
- Wilson, Thomas, heart disease complicating fibroids, quoted, *Obst.* 215
- Wilson and Maccarty, relation between ulcer and cancer of stomach from histological point of view, quoted, *Surg.* 137
- Wimshurst type of influence machines, *Electr.* 73, 74
- Winchester, isolated outbreak of diarrhoea at, *Epid.* 97, 98
- Winds prevalent at Naples, *Baln.* 19, 20
- WINGRAVE, V. WYATT.—Microscopical report on case of endothelioma of the ethmoid described by C. Nourse, *Laryng.* 63
 —, notes on the pathogeny of cholesteatomata, *Otol.* 63; discussion (p. 68): Dr. D. Grant, Dr. Milligan, Dr. Pritchard, Mr. Scott, Mr. West, Dr. D. McKenzie, Dr. Wingrave (reply)

- Winslow's soothing syrup, poisonous proprietary medicine, *Therap.* 89
- Winter station, Naples as, *Baln.* 27
- Wisdom teeth, impacted, causing neuralgia (C. Schelling), *Odont.* 114
- Women, angioneurosis only seen in, *Clin.* 107
- , gonorrhoeal arthritis in, *Baln.* 111, 112
- , less resistant to cosmic influences than men, *Baln.* 2
- , toleration of pain by, marked, *Obst.* 3
- WOOD, G., and WILSON, S. A. K.—Two cases of a nervous disease of undetermined nature occurring in a brother and sister, *Neur.* 96; discussion (p. 98): Dr. H. Head, Dr. S. A. K. Wilson (reply)
- Woodhead, Professor G. Sims, channels of tuberculous infection, quoted, *Child.* 27
- WOODS, R. S., TIDY, H. L., and ZUNDEL, C. E.—Statistics of pericardial effusion, from the London Hospital, *Med.* 115
- WOODWARD, IVY E.—Statistics of pericardial effusion, from Royal Free Hospital, *Med.* 114
- WOOLCOCK, W. J.—Discussion on proprietary, patent, and secret remedies, *Therap.* 96
- Woolwich, reduction of death-rate from diarrhoea in, *Epid.* 126
- Working classes, duty of Board of Education to, *Epid.* 40
- , enforcement of duties of mothers to children among, *Epid.* 43
- , prevention of improvident and unhealthy marriage-unions among, *Epid.* 43
- , weekly expenditure on alcohol among, *Epid.* 9
- Working men, cause of intemperance among, *Epid.* 41
- , hygienic importance of prevention of idleness among, *Epid.* 42
- Workington, outbreak of typhoid fever at, *Epid.* 261
- Workmen's Compensation Act, *Epid.* 37
- WRIGHT, Sir A. E.—Discussion on treatment of inoperable sarcoma by mixed toxins, *Surg.* 46
- , on opsonins, quoted, *Odont.* 32
- , discussion on vaccine therapy, *R.S.M.* 1
- WRIGHT, G.—Discussion on cholesterol gall stones containing typhoid bacilli, *Clin.* 171
- Wycombe, medical history of, *Baln.* 43
- WYLIE, A.—Case of obstruction of both nostrils, *Laryng.* 149; discussion (p. 149): Dr. D. Grant, Dr. Westmacott, Dr. Wylie (reply)
- , discussion on case of "singer's node" on left cord, *Laryng.* 108
- , leukoplakia (?) of the soft palate, *Laryng.* 82; discussion (p. 83): Dr. Dundas Grant, Dr. Donelan, Dr. Wylie (reply)
- , papilloma of the larynx, with symmetrical papillomata of the palate, *Laryng.* 95; discussion (p. 95): Dr. Jobson Horne, Dr. Wylie (reply)
- , tuberculosis of pharynx, *Laryng.* 58; discussion (p. 59): Mr. Hett, Dr. StClair Thomson, Dr. Scanes Spicer, Dr. Jobson Horne, Dr. Fitzgerald Powell
- WYNTER, W. ESSEX.—Case of angioneurosis, *Clin.* 105; discussion (p. 106): Dr. Wynter, Dr. Galloway, Dr. F. Parkes Weber, Dr. Rolleston, Dr. Wynter (reply)
- , case of chloroma, *Child.* 18; discussion (p. 19): Dr. Wynter, Dr. Sutherland, Dr. Whipham, Dr. E. I. Spriggs, Mr. Sydney Stephenson, Dr. Wynter (reply)
- , the diagnosis of aneurysm of the descending thoracic aorta, *Clin.* 125-127; discussion (p. 128): Dr. Alexander Morison, Mr. Pearce Gould, Dr. Wynter (reply)
- , discussion on case of congenital œdema of leg, *Child.* 89
- , on stricture of the rectum treated by fibrolysin, *Clin.* 90
- , sequel to a case of chloroma, *Child.* 96; discussion (p. 97): Dr. Ledingham, Dr. Wynter (reply)
- Xantho-erythrodermia perstans, case for diagnosis (G. W. Dawson), *Derm.* 27
- Xanthoma tuberosum multiplex (J. M. H. MacLeod), *Derm.* 108
- Xeroderma pigmentosa, case with unusual features (W. H. Evans), *Derm.* 57
- X-ray carcinoma, case of (J. Howell Evans), *Clin.* 145
- , dermatitis, chronic, treatment by refrigeration, *Electr.* 44

- X-ray dermatitis, treatment by vaccine, R.S.M. 14
— stereoscope, simple (A. J. H. Iles), Electr. 138
— tube, application of electromotive force to, Electr. 151
— —, effect of Snook's high-tension transformer on, Electr. 158
— — — — —, see also *Röntgen rays*
X-rays, see *Röntgen rays*

- YEARSLEY, MACLEOD.—Discussion on large nasopharyngeal growth in a boy aged 12, Otol. 48
— — on pathological changes found in ear of deaf-mutes, Otol. 13
—, discussion on pedunculated papilliform growth, Otol. 46
Yeo, I. Burney, oro-nasal inhaler, quoted, Therap. 18, 19
York, poverty in, method of estimation by Mr. Rowntree, Epid. 4, 12, 19
Youths, uselessness of unskilled labour to, Epid. 38

- ZUNDEL, C. E., TIDY, H. L., and WOODS, R. S.—Statistics of pericardial effusion, from the
London Hospital, Med. 115

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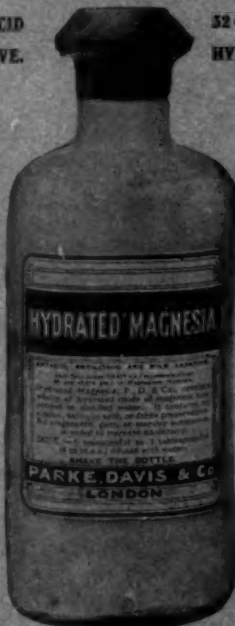
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CONTENTS

	PAGE
Balneological and Climatological Section.	
To Redress the Balance (Presidential Address). By LEONARD WILLIAMS, M.D.	1
The Hygiology of Naples. By JOHN J. EYRE	14
Section for the Study of Disease in Children.	
Cases of Subacute Liver Atrophy, persistent Vomiting, and Lymphosarcoma, with Notes on Liver Pathology in Childhood. By A. DINGWALL FORDYCE, M.D.	1
Pneumococcal Infection in an Infant simulating Generalized Tuberculosis. By WALTER CARR, M.D.	14
Specimens from a Case of Lymphosarcomatosis, with Clinical and Post-mortem Notes. By F. W. HIGGS, M.B.	17
Chloroma. By W. ESSEX WYNTER, M.D.... ..	18
Two Cases of Acute Anterior Poliomyelitis involving the Four Limbs. By A. MANUEL, M.B.	21
Extracapsular Fracture of Neck of Thigh-bone in Boy aged 6. By RALPH THOMPSON, F.R.C.S.	22
Congenital Hemidystrophy. By J. HOWELL EVANS, M.Ch.	23
Case for Diagnosis. By J. P. LOCKHART MUMMERY, F.R.C.S.	24
Acute Inflammation in an Appendix, involuted into the Lumen of the Cæcum, which then formed the Apex of an Intussusception; Operation; Recovery. By IVOR BACK, F.R.C.S.	25
Tuberculosis of the Tonsil, associated with Tuberculous Glands of Neck. By E. SCOTT CARMICHAEL, F.R.C.S.Ed.	27
Clinical Section.	
Further Report on a Case of Carcinoma of the Breast treated by Radium. By N. S. FINZI, M.B., and ERNEST H. SHAW	1
Demonstration by the Ultra-microscope of living <i>Treponema pallidum</i> and various Spirochaetes. By H. W. BAYLY	3
Mycosis Fungoides. By J. L. BUNCH, M.D.	6
Pigmentation of the Circum-oral Skin and of the Buccal Mucosa in Pernicious Anemia. By H. D. ROLLESTON, M.D.	9
Excision of both Maxillæ. By ALBERT CARLESS, M.S.	10
Meckel's Diverticulum containing Calculi and producing Colic. By JAMES SHERREN, F.R.C.S.	11
Rheumatoid Arthritis with Enlargement of Lymphatic Glands and Spleen. By E. I. SPRIGGS, M.D.	15
Flexor Spasm. By G. A. SUTHERLAND, M.D.	19
Detachment of the Tendon of the Subscapularis Muscle for Recurring Dislocation of the Humerus. By W. G. SPENCER, M.S.	20

Schlatter's Disease. By GORDON TAYLOR, M.S.	20
By Sir DYCE DUCKWORTH, Bt., M.D. :—	
(1) Gout, with large Tophaceous Deposits, in a Mahommedan ...	21
(2) Gout, with Uratic Tophi, in a Boy aged 14	23
By W. P. HERRINGHAM, M.D.	
(1) Two Cases of Lobar Pneumonia, with considerable Displacement of the Heart	25
(2) Further Notes on a Case of Neuritis of Brachial Plexus ...	28

Dermatological Section.

By H. G. ADAMSON, M.D. :—	
(1) Multiple Scars simulating Leucoderma Syphiliticum	1
(2) Small Follicular Syphilide of the Corymbose Type	1
By E. G. GRAHAM LITTLE, M.D. :—	
(1) Lichen Scrofulosorum	2
(2) Acute and very Extensive Eruption of Erythema Iris Type in a Man aged about 35	2
(3) Urticaria Pigmentosa of very Exceptional Severity	3
Cystic Rodent Ulcer. By J. M. H. MACLEOD, M.D.	3
By Sir MALCOLM MORRIS, K.C.V.O., F.R.C.S.Ed., and S. E. DORE, M.D. :—	
(1) Thickening of the Skin of the Palms and Soles in a Woman aged 31 ...	6
(2) Curious Affection of the Finger- and Toe-nails	6
(3) Report on Case of Extensive Acne with Scars	7
A Series of Cases of Scrofuloderma and Lupus treated by Tuberculin Injections. By G. T. WESTERN, M.D.	8
Case for Diagnosis. By W. KENNETH WILLS, M.B.... ..	12

Electro-Therapeutical Section.

Electro-therapeutics in Gynæcology (Presidential Address). By SAMUEL SLOAN, M.D.	1
---	---

Epidemiological Section.

Poverty and Disease (Presidential Address). By JAMES NIVEN, M.B.... ..	1
--	---

Medical Section.

On Oxaluria and the Treatment of Calcium Oxalate Deposit from the Urine, with a Method for the Solution of Calcium Oxalate Calculus whilst in the Urinary Passages. By ROBERT MAGUIRE, M.D.	1
--	---

Obstetrical and Gynæcological Section.

Pain associated with Disorders of the Female Genital Organs (Presidential Address). By H. MACNAUGHTON-JONES, M.D.	1
Placenta Prævia in which it was deemed advisable to perform Caesarean Section. By J. M. MUNRO KERR, M.D.	11
Points for the Use of, and Indications for, Electrotherapy in some Gynæcological Affections. By J. CURTIS WEBB, M.B.	16

Odontological Section.

The Relation of Dentistry to other Branches of Medicine and its Bearing upon the Public Health (Presidential Address). By WILLIAM HERN, M.R.C.S., L.D.S.	1
Infective Disease of the Jaws associated with the absorption of the Teeth. By A. HOPEWELL-SMITH, M.R.C.S., L.D.S.... ..	9
Papillomata of the Uvula. By F. COLEMAN, M.R.C.S., L.D.S.	20

Pathological Section.

The Present Position of the Neurone Doctrine in relation to Neuro-pathology (Presidential Address). By F. W. MOTT, F.R.S.	1
Rhabdomyoma of the Urinary Bladder. By S. G. SHATTOCK	31
A Note on Eosinophile Cells in the Exudate from Tick-bites on a Horse. By J. BURTON CLELAND	42

Surgical Section.

The Treatment of Inoperable Sarcoma by Bacterial Toxins (the Mixed Toxins of the Streptococcus erysipelas and the Bacillus prodigiosus). By WILLIAM B. COLEY, M.D. (New York)	1
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A Discussion on Teaching of the Therapeutics in the Hospital Wards. Opened by Sir T. CLIFFORD ALLBUTT, K.C.B., M.D., F.R.S.	1
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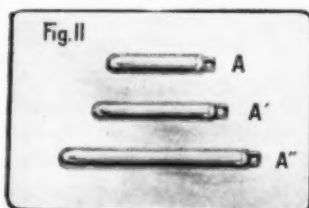
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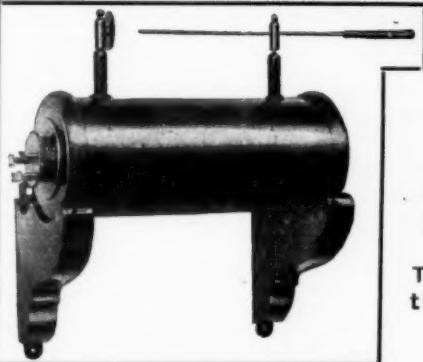
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